SPECIAL ISSUE OF

THE

LARYNGOSCOPE.

VOL. LXVIII MARCH, 1958 No. 3

PROCEEDINGS OF THE INTERNATIONAL CONFERENCE ON AUDIOLOGY

May 13 through 16, 1957.

St. Louis, Missouri.

FOREWORD.

In connection with the official Midwest tours scheduled for the Sixth International Congress of Otolaryngology, held in Washington, May 5-10, 1957, one of the Organizing Committee, (S. R. Silverman) was asked by numerous individuals, both in the United States and abroad, to organize an International Conference on Audiology in St. Louis immediately after the Congress. The chief purpose of this Conference would be to create a situation where laboratory investigators could share their views and findings with an interested and sophisticated clinical audience. The themes explored by the Conference were the Assessment of Auditory Function; the Physiology of Audition, and the Relation of Hearing Loss to Noise Exposure.

The goals of the Conference are summarized in the follow-

ing extract from the welcoming address of the Chairman to the members of the Conference:

To Participants in the International Conference on Audiology—A Cordial Welcome to St. Louis:

From many lands a common interest in hearing and deafness brings us together. It is true that our primary interests may vary. We may be concerned with the structure of the auditory system or with its function, or with the diseases that afflict it, or with the impairment in social efficiency brought about by hearing loss. Some of us work in the laboratory, others in the clinic and still others in the classroom; but the rapidity with which our knowledge, nurtured by many sources, is increasing, suggests clearly and emphatically that we create situations, such as this Conference, where we may share information that enriches our understanding and stimulates our activity. If the Conference contributes in some small measure to the enthusiasm and the wisdom with which you pursue your tasks in the field of hearing and deafness, those responsible for its organization will have been richly rewarded.

* * *

Of the actual scientific and professional membership of the Conference, fields of interest were represented as follows: Audiology, including teachers of the deaf and speech pathologists, 42 per cent; Otolaryngology, 39 per cent; Psychology, Engineering and Physiology, 15 per cent; and Commercial, including makers of electroacoustic instruments, 4 per cent.

The editors of the Proceedings of the International Conference on Audiology were S. Richard Silverman, Chairman of the Conference; Ira J. Hirsh, Program Chairman; and Shirley K. Hirsh, Conference Secretary.

The Conference acknowledges with appreciation its indebtedness to the National Science Foundation of the United States Government for providing the major portion of its expenses through a grant to the Central Institute for the Deaf.

The Conference expresses its gratitude to the members of the staffs and friends of the Central Institute for the Deaf, and the Department of Otolaryngology of Washington University for assistance in arrangements; and to the School of Medicine of Washington University for provision of meeting rooms.

PROGRAM OF TECHNICAL SESSIONS.

Tuesday Morning, May 14.

WELCOME:

Presiding: S. Richard Silverman, Director, Central Institute for the Deaf; Chairman, International Conference on Audiology.

The Honorable Raymond R. Tucker, Mayor of the City of St. Louis.

Ethan A. H. Shepley, Chancellor, Washington University.

SESSION A—CONTRIBUTED PAPERS:

Chairman: Ira J. Hirsh, Head, Psychology Laboratory, Central Institute for the Deaf, St. Louis, Mo.

1. **Some Similarities Between Hearing and Seeing.**

   S. S. Stevens, Professor of Psychology, Director of Psycho-Acoustic Laboratory, Harvard University, Cambridge, Mass..... 508

2. **An Attempted Synthesis of Psycho-Acoustic Test Data in the Audiological Clinic.**

   J. Donald Harris, Head, Sound Branch Medical Research Laboratory, U. S. Naval Submarine Base, New London, Conn..... 527

3. **Triplet Testing and Training—An Approach to Band Discrimination and Its Monaural or Binaural Summation.**

   Henk C. Huizing, Professor of Audiology, University of Groningen, The Netherlands, and M. Taselaar, University of Groningen 535

4. **Relation of the Lombard Effect to Speech Production.**

   John J. Dreher and John J. O'Neill, Psycho-Linguistics Laboratory, Ohio State University Research Foundation, Columbus, Ohio 539

5. **A Comparison of Picture Response and Hand Raising Techniques in Pure Tone Audiometry with Young Children.**

   Harriet Kaplan, Graduate Assistant, and Bruce M. Siegenthaler, Associate Professor of Clinical Speech, Pennsylvania State University, University Park, Pa..... 548

6. **Progress in the Comparison of Sleep EEG Audiometry with Standard Audiometry.**

   A. J. Derbyshire, Director EEG Department, Harper Hospital, Detroit, Mich., and

   Mary McDermott, Head, Outpatient Services, Children's Hospital of Michigan, Detroit, Mich..... 558

Tuesday Afternoon, May 14.

ASSESSMENT OF AUDITORY FUNCTION:

I. PANEL DISCUSSION—HEARING IN CHILDREN

Moderator: S. RICHARD SILVERMAN, Director, Central Institute for the Deaf, St. Louis, Mo. 218

Panel Members: ROBERT GOLDSTEIN, Research Associate, Central Institute for the Deaf, St. Louis, Mo.

WILLIAM G. HARDY, Director, Hearing and Speech Center, The Johns Hopkins Hospital, Baltimore, Md.

HELMER R. MYKLEBUST, Professor of Audiology, Northwestern University, Evanston, Ill.

LOUISE TRENQUE, Chief, Audiometric Department, University of Lyons, France.

II. DIAGNOSIS OF HEARING DISORDERS

Chairman: IRA J. HIRSH, Head, Psychology Laboratory, Central Institute for the Deaf, St. Louis, Mo.

Audiometry in Diagnosis:

RAYMOND CARHART, Professor of Audiology, Northwestern University, Evanston, Ill. 253

Ménière's Disease:

THEODORE E. WALSH, Professor of Otolaryngology, Washington University School of Medicine, St. Louis, Mo. 279

Cochlear vs. Retrocochlear Lesions:

TAUNO PALVA, Associate Professor of Clinical Audiology, University of Turku, Finland 288

Clinical Aspects of Cortical Deafness:

ETTORE BOCCA, Chief, ENT Clinic, University of Milan, Italy..... 301

Tuesday Evening, May 14.

Open House—Central Institute for the Deaf, Clinics and Research Building.

Demonstration of classroom procedures with deaf, hard-of-hearing, and aphasic children, and also of testing and teaching techniques for children and adults.

Research laboratories and shops were open for inspection and demonstration.

Exhibitions included Central Institute Library and the Goldstein Collection of hearing aids.

A film by Georg von Békésy was shown at 8:30 and 9:30 P.M.

Synopsis of Békésy film "Pendulums, Traveling Waves, and the Cochlea" 317

Wednesday Morning, May 15.

PHYSIOLOGY OF HEARING:

Chairman: HARVEY FLETCHER, Director of Research, Brigham Young University, Provo, Utah.

I. PROCESS OF SOUND CONDUCTION:

MERLE LAWRENCE, Associate Professor of Physiological Acoustics, University of Michigan Medical School, Ann Arbor, Mich..... 328

Technical Discussion:

HENNING E. VON GIERKE, Chief, Bio-Acoustics Branch, Aero Medical Laboratory, United States Air Force, Wright Air Development Center, Dayton, O. 347

Clinical Discussion:

TERENCE E. CAWTHORNE, London, England 354

Film:

HEINRICH G. KOBRAK, Professor of Otologic Research, Wayne University College of Medicine, Detroit, Mich. 359

II. TRANSMISSION AND TRANSDUCTION IN THE COCHLEA:

HALLOWELL DAVIS, Director of Research, Central Institute for the Deaf, St. Louis, Mo. 359

Theoretical Discussion:

J. C. R. LICKLIDER, Associate Professor of Psychology, Massachusetts Institute of Technology, Cambridge, Mass. 382

Clinical Discussion:

JOHN R. LINDSAY, Professor of Otolaryngology, University of Chicago, Chicago, Ill. 385

Wednesday Afternoon, May 15.

Chairman: JOSEPH ERLANGER, Nobel Laureate and Professor Emeritus of Physiology, Washington University School of Medicine, St. Louis, Mo.

III. TRANSMISSION IN THE CENTRAL NERVOUS SYSTEM:

ROBERT GALAMBOS, Chief, Department of Neurophysiology, Walter Reed Army Institute of Research, Washington, D. C..... 388

Physiological Discussion:

ICHIJI TASAKI, Chief, Special Senses Section, Laboratory of Neurophysiology, National Institute of Neurological Diseases and Blindness, Bethesda, Md. 401

Anatomical Discussion:

GRANT L. RASMUSSEN, Chief, Section on Functional Neuroanatomy, National Institute of Neurological Diseases and Blindness, Bethesda, Md. 404

Clinical Discussion:

J. S. RIESCO-MACCLURE, Neuro-Surgical Institute, Santiago, Chile 406

IV. FUNCTION OF THE AUDITORY CORTEX:

W. DEWEY NEFF, Professor of Psychology, University of Chicago, Chicago, Ill. 413

Physiological Discussion:

ARCHIE R. TUNTURI, Assistant Professor of Anatomy, University of Oregon Medical School, Portland, Ore. 419

Clinical Discussion (Neurosurgery):

ROBERT B. KING, Assistant Professor of Neurological Surgery, Washington University School of Medicine, St. Louis, Mo. 424

Clinical Discussion (Otology):

HAROLD F. SCHUKNECHT, Associate Surgeon, Division of Otolaryngology, Henry Ford Hospital, Detroit, Mich. 429

Thursday Morning, May 16.

HEARING LOSS AND NOISE EXPOSURE:

Chairman: WALTER ROSENBLITH, Massachusetts Institute of Technology, Cambridge, Mass.

I. NOISE IN INDUSTRY:

JEROME R. COX, JR., Head, Acoustics Laboratory, Central Institute for the Deaf, St. Louis, Mo. 440

II. HEARING LOSS IN INDUSTRY:

ARAM GLORIG, Director of Research, Subcommittee on Noise in Industry, American Academy of Ophthalmology and Otolaryngology, Los Angeles, Calif. 447

III. A LABORATORY METHOD FOR THE STUDY OF ACOUSTIC TRAUMA:

DONALD H. ELDREDGE, Research Associate, Central Institute for the Deaf, St. Louis, Mo.

WALTER P. COVELL, Associate Professor of Anatomy and Otolaryngology, Washington University School of Medicine, St. Louis, Mo. 465

IV. DIAGNOSIS, SUSCEPTIBILITY AND PRESBYCUSIS:

GORDON HOOPLE, Professor of Otolaryngology, University of the State of New York Medical School, Syracuse, N. Y. 477

V. EAR PROTECTION:

JOSEPH ZWISLOCKI, Research Fellow, Psycho-Acoustic Laboratory, Harvard University, Cambridge, Mass. 486

VI. THE ESTABLISHMENT OF CRITERIA BASED ON THE CONCEPT OF NOISE EXPOSURE:

WALTER A. ROSENBLITH, Associate Professor of Communications Biophysics, Massachusetts Institute of Technology, Cambridge, Mass. 497

Thursday Afternoon, May 16.

SESSION B—CONTRIBUTED PAPERS:

Chairman: LENNART GISSELSON, Assistant Professor, Otolaryngology, University of Lund, Sweden.

1. **Behavior of the Neuroglia of the Acoustic Nuclei, Sub-cortical Centers and Auditory Cortex of Cats Intoxicated with Dihydrostreptomycin.**
GERALDO DE SA, Assistant Professor of Otolaryngology, University of Recife, Brazil 571

2. **Observations and Considerations About the Cochlear Innervation of the Cat.**
PAOLO CONTU, Head Professor of Anatomy, University of Recife, Brazil 586

3. **DC Potentials of the Membranous Labyrinth.**
CATHERINE A. SMITH and C. F. GESSERT, Department of Otolaryngology, Washington University School of Medicine, St. Louis, Mo.;
H. DAVIS and B. H. DEATHERAGE, Central Institute for the Deaf, St. Louis, Mo. 595

4. **The Modifications of Cochlear Potentials Produced by Streptomycin Poisoning and by Chronic Stagnant Anoxia.**
CESAR FERNANDEZ and R. S. KIMURA, Division of Otolaryngology, University of Chicago, Ill.;
C. A. SMITH, Department of Otolaryngology, Washington University School of Medicine, St. Louis, Mo.;
B. ROSENBLUT, B. H. DEATHERAGE and H. DAVIS, Central Institute for the Deaf, St. Louis, Mo. 596

5. **Acetylocholinesterase and the Olivo-Cochlear Tract of Rasmussen.**
HAROLD F. SCHUKNECHT, Associate Surgeon, Division of Otolaryngology, Henry Ford Hospital, Detroit, Mich. 627

6. **Response to Auditory Clicks of the Neurally Isolated Cochlea of the Cat.**
KARL LOWY and ARNOLD A. GERALL, Research Associates, Department of Psychology, University of Rochester, N. Y. 628

SESSION C—CONTRIBUTED PAPERS:

Chairman: VICTOR GOODHILL, Associate Clinical Professor of Otolaryngology, University of Southern California, Los Angeles, Calif.

1. **Applications and Limitations of Auditory Tests in the Selection of Cases for Stapes Mobilization.**
SAMUEL ROSEN, Consulting Otologist and Head, Stapes Mobilization Clinic, Mt. Sinai Hospital, New York, N. Y.; and
MOE BERGMAN, Director, Hearing and Speech Center, Hunter College, New York, N. Y. 629

2. Measurement of Stapes Fixation.
Otto H. Meurman, Docent of Otolaryngology and Director of Audiology Center, University of Helsinki, Finland 630

3. Direct Audiometry in Audiosurgery.
Francisco Antoli-Candela, Chief, Audiology and Audiosurgery Service, National Institute of Medicine and Security of Work; and Fernando Antoli-Candela, F. Olaizola, L. Saft, Madrid, Spain 635

4. New Approach to the Incudo-Stapedial Region for Mobilization of the Stapes.
Luis Garcia-Ibanez, Otorhinolaryngologist, National Health Service, Valencia, Spain 636

5. The Carhart-Shambaugh Formula Used for Stapes Mobilization Prediction.
Frederick R. Guilford and C. Olaf Haug, Houston, Texas..... 637

6. Recent Studies on Occupational Deafness and Auditory Fatigue.
H. A. V. von Dishoeck, Professor of Oto-Rhino-Laryngology, Leyden University, The Netherlands 645

7. Temporary Hearing Loss at 4000 c.p.s. as a Function of the Intensity of a Three-Minute Exposure to a Noise of Uniform Spectrum Level.
James D. Miller, Research Associate, Hearing and Communication Laboratory, Indiana University, Bloomington, Ind. 660

8. Clinical Bone-Conduction Audiometry at the Frontal Bone.
Ralph F. Naunton, Instructor, Department of Otolaryngology, University of Chicago, Ill. 671

9. Proposed Audiology Centre of the National Centre for the Deaf in India.
C. A. Amesur, Member, National Advisory Council for the Education of the Handicapped, Bombay, India; and
C. Satyanarayana, Professor of E.N.T. Diseases, Stanley Medical College, Madras, India 675

NATIONAL ORGANIZING COMMITTEE.

Chairman:

S. R. Silverman, Central Institute for the Deaf, and Washington University.

Raymond Carhart, Northwestern University.

Hallowell Davis, Central Institute for the Deaf, and Washington University School of Medicine.

Harvey Fletcher, Brigham Young University.

William G. Hardy, Johns Hopkins Hospital.

John Lindsay, University of Chicago.

Philip E. Meltzer, Tufts University Medical School.

S. S. Stevens, Harvard University.

Program Chairman:

Ira J. Hirsh, Central Institute for the Deaf, and Washington University.

Conference Secretary:

Mrs. Shirley K. Hirsh.

TUESDAY AFTERNOON SESSION,

May 14, 1957.

THE ASSESSMENT OF AUDITORY FUNCTION.

I. HEARING IN CHILDREN—PANEL DISCUSSION.

CHAIRMAN: S. Richard Silverman, Director, Central Institute for the Deaf, St. Louis, Mo.

PANEL: Robert Goldstein, Research Associate, Central Institute for the Deaf, St. Louis, Mo.
William G. Hardy, Director, Hearing and Speech Center, Johns Hopkins Hospital, Baltimore, Md.
Helmer R. Myklebust, Professor of Audiology, Northwestern University, Evanston, Ill.
Louise Trenque, Chief, Audiometric Department, University of Lyons, France.

DR. SILVERMAN: The essayists were asked three questions in preparation for this discussion. These questions are as follows:

One: What goals, particularly with reference to educational recommendations, can be set for the testing of the hearing of young children?

Two: What evidence is there that results of hearing tests on young children can yield information about the pathology of auditory and other disorders?

Three: What particular testing techniques have you found to be most successful in yielding answers to either or both of the first two questions?

One, then, is a matter of goals; two, is a matter of evidence; and three, what do you actually do when you assess the hearing of a young child?

Dr. Hardy, will you lead off?

DR. WILLIAM G. HARDY: Any one of these topics is worth

an hour's discussion, so I am going to move through my material rather rapidly and hope that we may be able to pick up some of the more important indicators as the discussion proceeds.

There are at least six objectives which we feel (I am talking as a representative of our departmental group) might well be kept in mind. First, to ascertain the status and needs of the child regarding possible medical and surgical treatment. This aspect of a problem is too often overlooked, and it must not be, particularly in dealing with younger children. Incidentally, need we remind ourselves that we are not talking about a refined, preselected diagnostic group, all of whose problems are solely centered in hearing? The children we meet at the age of one, two, or three years, are referred because they do not talk; because they have not learned language; because they have not developed normally. The reasons for these defects are manifold, involving much more than the possibility of impaired hearing.

Second, to measure or estimate the child's potential for learning language and verbal communication. Fundamental to this, perhaps, is the potential of the auditory mechanism, but by no manner of means should the inquiry exclude other sensory modes of learning.

Third, to differentiate among several possible reasons for the lack of development of language and speech. In terms of differential diagnosis, this may involve a tremendously complex inquiry. We find that oftentimes this involves a closely integrated task among five or six of our major departments in the hospital.

Fourth, to measure residual hearing as a basis for psychoacoustic development.

Fifth, to try to learn the nature of the hearing impairment for what this information can contribute in terms of special needs in training. Many of these objectives overlap, of course, and any effort to clarify reflects some of the comments in another paper given this morning: our need to clarify our language in terms of what we mean by *hearing*. Is hearing an "adequate response," or is it a "significant

response" to a measured or variable set of stimuli, or is it simply "a response." Our references must be clarified, for in his experience with differential diagnosis one comes face to face very early with the realization that he inevitably is dealing with much more than the function of the auditory system. The auditory system presents only one kind of information to the mind. As one watches more and more of these tiny children who are apt to give sporadic, variable, inconsistent responses to sound, one is forced to wonder what this auditory experience is like to the child. How can it be described in a single concept? Suppose that a given child has some disorder or lack of development at the level of difficulty in brief memory-span. This might be directly, or only remotely, connected with an auditory deficit; yet what would the world of sound-stimulus, particularly speech-stimulus, be like to a little fellow who needs 75 milliseconds, instead of five milliseconds, in order to monitor his own vocal output? These are points of differentiation on which we have as yet barely begun to touch. The modes of trying to apprehend, measure, and describe this sort of deficit are still in their infancy. Related, without doubt, are variables in pitch-perception, perhaps at the cortical or immediately subcortical levels of the auditory mechanism.

Sixth, to develop information relative to the nature of the hearing mind in terms of whatever effects of stimulus and inhibition can be observed in the responses of the child. A child must go through a period of readiness to listen before he can progress very far in the development of language and speech. This is, in part, a matter of the mind's meanings, of the conditioning of behavior to react or not to react to a particular sensory modality-like hearing. It is probably at this stage of clinical judgment that one must differentiate between the fact of a child's response to sound, and a possible meaning of this experience to him. Here we are at the level of cortical integration, and there simply is not time to discuss the many possible ramifications.

I believe that all of these become extremely important objectives in an effort to refine hearing tests with reference to educational recommendations.

Dr. Hirsh, translating for Dr. Louise Trenque:*

DR. IRA J. HIRSH: There are four major objectives: one, is the child deaf? In the young child, in the majority of cases, the problem consists of establishing primarily whether or not deafness exists. The fact that the child is helped most by the specialist from about 18 months on is because the language either does not appear or because the language is not developed normally. Frequently the parents suspect the existence of deafness, but it is still necessary to confirm it.

The second objective is to quantify the extent of the deafness. Is deafness complete? Are there existing auditory residuals that might be used by the child in hearing speech efficiently? The value of these auditory residuals is most important if it appears that the child can hear speech partially, or possibly better with a hearing aid. By using these residuals, for example, in perceiving some of the phonemes or the melody of the sentence, one can hope to rehabilitate the child completely. Thanks to appropriate education this child can become capable of speaking with a voice that is comprehensible to all, capable of understanding what is said to him, and capable of acquiring even abstract thought at a level consistent with children of the same intellectual capacity and environment.

Third, is deafness the only deficiency in the child? In our opinion it is of prognostic importance to ascertain whether the deafness is the only trouble, or whether there are others such as physical characteristics, deficiency in intelligence, difficulty in memorization and comprehension, superimposed upon the auditory deficit. A large proportion of children in whom we have found audiometric curves of between 60 and 80 decibels, in whom, because of these auditory residuals, we have placed much hope, have disappointed us following the examinations. In the course of their education, we were

*Dr. Louise Trenque was unable to attend the Conference because of illness. She sent a manuscript, however, through one of the conferees, which was received about an hour before the panel discussion was to begin. Dr. Hirsh's translation of her answers to the questions posed by the Chairman was therefore almost in the nature of a simultaneous translation. There are many omissions and undoubtedly some errors, but the Editors consider that at least her general point of view is fairly represented.

confronted with various difficulties, among the most severe of which have been dyslexia, dysgraphia, emotional instability, disorders in the development of learning, comprehension and intelligence, and those factors that interfere with the development of language beside those involved in the hearing.

Finally, as the fourth objective, Dr. Trenque lists the study of the origin of deafness. This study has both therapeutic and prophylactic aims and can aid in our prognostic judgments: therapy in cases of rhinogenic deafness; prophylaxis, for example, in the RH factor or syphilis; prognosis, as in cases where we observe obstetrical trauma.

DR. ROBERT GOLDSTEIN: Because our concern on this panel is primarily educational, we will consider the results of hearing tests as one basis for making educational recommendations. My discussion today is restricted, first, to those auditory problems whose severity requires that education be handled, at least for a time, in special schools or special classes; two, to the very young children who have not yet had any individual or classroom instruction, from which we might ordinarily derive significant diagnostic clues; and third, to those children whose auditory disorder is not secondary to severe emotional disturbance or to mental deficiency.

Our goals should depend primarily on what we know of the kinds of educational methods that have been used successfully on children with various auditory disorders. My discussion is further restricted to what is known in general about the success of various approaches to oral education, acknowledging our desire to accord every child the opportunity for developing oral communication.

Considering the present state of knowledge about the education of children with auditory disorders, I believe that we should find out from our hearing tests whether a child's auditory disorder results primarily from a serious reduction in auditory sensitivity or primarily from a defect in auditory perception and linguistic development.

The education of hard-of-hearing children and deaf children, that is, children with varying losses of auditory sensitivity, although not uniform or simple, has been carried on

with appreciable success, and has been described in some detail. By comparison, we know very little about the success in educating children whose disorder can be described as defective auditory perception, or as the inability to produce and/or to understand language. There are scattered reports of success with certain educational approaches, but there are no comprehensive reports in which an educational method has been described, the children taught by this method clearly defined and enumerated, and careful assessment of results presented. I feel competent, therefore, to refer only to our own experience for my contribution to the evaluation of this question.

We have classified a large number of our children as aphasic. We are aware of the controversies around this term, and for now, at least, we are not restricting ourselves to any particular definition of it. These children we call aphasic have as their main problem the inability to develop language, presumably because of some dysfunction of the central nervous system. This is not a homogeneous group, either from the standpoint of organic problems or from the standpoint of ability to use language; nevertheless, we use the same educational method with all of these children and achieve varying but considerable success with them. We make no attempt to develop a different procedure for each sub-classification of the aphasic group but modify certain aspects of our overall procedure when the situation calls for it.

If the findings on a particular child are inconsistent and do not clearly identify a child as deaf or aphasic, we generally recommend that this child be taught according to the system we use with aphasic children. We have found from experience that we do little educational harm to children who eventually prove to be simply deaf, by teaching them according to our system for aphasic children. On the other hand, we have seen much educational time lost on aphasic children who, because of uncertainty in diagnosis, were taught by methods conventionally used with deaf children.

On the basis, then, of our experience in educating children with auditory disorders, we try to answer this major question when we are examining a young child: is his inability to

communicate primarily the result of a reduction in auditory sensitivity or is it primarily the result of a defect in auditory perception or in the ability to produce or to understand language?

DR. SILVERMAN: We turn now to our second question. What evidence is there that results of hearing tests on young children can yield information about the pathology of auditory or other disorders?

DR. WILLIAM G. HARDY: I seem to have ten points listed for this topic. First, it is clear that with the use of a wide variety of techniques (and each of us selects the particular pattern that he finds most rewarding) the function of the peripheral hearing mechanism—the end-organ—can be measured. There is a matter of definition here that is apt to make discussion confusing. Where does the peripheral mechanism stop and the central mechanism begin? My group has given a great deal of thought to this question; it is not simple. Let us assume that the central mechanism begins at the level of the brain stem where one is dealing with audition as a neurally bilateral sequence of events, and that the ganglion-cell structure may be considered in the peripheral mechanism. I realize there may be something wrong with this, for we are including the first neural synapse in the end-organ. The geography of anatomy is perhaps not quite so important, however, as an agreement on reference. In terms of these distinctions between peripheral and central, I believe we have a right to say that there is incontrovertible evidence that the function of the peripheral mechanism can be measured quite adequately.

From here on, a clear differentiation of possible pathology is in large part speculative. Fortunately, in this regard, one has the opportunity (which he must learn to take) of follow-up, or reassessment; as Dr. Goldstein just indicated, to take advantage of comments and observations from the teacher, derived from the child's day-to-day performance. A child is exposed to an ordered process of training in class, and it helps a great deal to know what a trained, thoughtful observer sees the child do. This kind of information is not available for a two-year-old, of course, but with ever younger age-ranges

being admitted these days to special educational situations, the evidence begins to mount up by the time a child is six or seven years old.

For many years in our department we have been particularly interested in the use and misuse of galvanic audiometry. We find that there are three kinds of patterns relating to the situation: *1.* the ease and speed of conditioning; *2.* the latency of the response; *3.* various details of timing involved in the test-picture. With relatively few exceptions, when one is dealing with damage to the peripheral mechanism, conditioning is easy, ready, and consistent. Latency of response is usually within half a second. The requirement of reinforcement may vary considerably. So, too, does the nature of the signal. Some children respond better to a steady-state tone, some better to a tone pulsed at a frequency of three or four per second. With the children whose primary problem turns out to be a language disorder, difficulty in integrating and putting to meaningful use the information presented through the auditory system, the same general patterns exist but with much extension of time. It takes longer for these children to be conditioned, and there may be extreme variations in latency of response. With the other large group of children who seem to have a central auditory problem, perhaps at the level of the interbrain or the thalamic loops, I doubt that the majority can ever be clearly conditioned. One may see responses, many of them, but they do not follow a pattern. There is much variety, too, in terms of latency and the details of extinction. This is perhaps relatable to the sort of thing currently being demonstrated in clinical psychology as more and more refinements of perceptive modes are studied in older children; some of these are relatable to various signs and symptoms of possible brain-damage, some are not. Psychologists use the term *variability* to describe many of the sensory inter-relations of the brain-damaged child, and it is felt that this is a good functional description of some of these central auditory problems. The signal, and the information derivable from it, changes, shifts. This is inconsistency from the point of view of the tester; variability from the point of view of the sub-

ject. One wonders what the world of sound is like under these circumstances.

When this discussion is finished we can probably all agree that the only really good test of the entire range of hearing, listening and understanding, the reflexes of the auditory mechanism and the integration of the cortical system, is a speech hearing test; but this, of course, remains beyond reach when one is dealing with small children, unless a particular child has developed some language and some useful speech.

Back again to the auditory system, there are many interesting observations to be made relative both to loudness and pitch, the two psychological parameters of auditory sensitivity. In our observations—and one doubts that the caseload we see is unusual—we find that among the children who offer clear evidence of profound auditory involvement, so much that they cannot possibly learn without the very best of special education, seven out of ten have something more than peripheral auditory lesions. In reference here is the preschool-age group. Approximately seven out of ten give evidence that their auditory difficulties lie somewhere in the brain stem or interbrain, in the thalamic structures, or in some aspect of cortical relationships.

Dr. Derbyshire made reference to a kind of "auditory hiatus" in some of these children. We have had similar experiences. Time and time again, we have tried to condition with a 90-decibel tone without success. Then we shift to a 25-decibel tone and immediately get responses. Sometimes, after a child begins to respond galvanically, one runs into a gap of perhaps 40 decibels; there are sporadic responses to various tones somewhere near the range of normal threshold acuity, but it requires a signal of 40 or so decibels above this level in order to achieve a consistent pattern of responses. This almost forces one to think of some of the auditory problems commonly seen in a child with Rh athetosis. Dr. Goodhill and others have demonstrated by histopathologic evidence that at least in some of these children the damage may well lie at the level of the auditory nuclei in the brain stem. We have examined many such problems, and find that with few

exceptions they want much more loudness via a hearing aid or other amplification than does a child or an adult with a comparable pure-tone curve representing a peripheral problem.

Some years ago, it became apparent that once there was good evidence of responses in the region of threshold acuity (we are still discussing galvanic skin resistance audiometry), it was profitable to observe reactions to a five- or six-octave pitch-swing at levels well above threshold. Some children respond to this stimulus, some do not. One cannot pretend to know the details of what this means. Where there is a consistent reaction to this changing acoustic event, however, one may assume that it represents an on-going, different event to the child. This capacity, we have observed, is commonly not present in children who prove to have extreme central auditory disorders, whether or not this is combined with an aphasoid problem.

On the other hand, there is a clear relation between awareness and intelligibility that can be studied, for example, in the problems of geriatric audiology. On much less secure bases, one can observe awareness of sound in a child (assuming that there is good evidence of responses within normal range), at much lower levels of intensity than can be used in terms of intelligibility. Many older children who have had the advantage of good training in language are aware of sound at much lower levels than they can put to use. It may be that a *listening* circuit is involved here.

One more point, a matter that we have found confusing. In the course of otologic and audiologic history we have all learned to use decibel notation in describing the intensity-loudness factors of an affected end-organ. One wonders, however, how anybody can make a statement in terms of decibel notation when the problem lies high in the central mechanism. The essence of this kind of problem is that it is not a pattern; it is not a constant; it does not remain "the same"; therefore, the idea of describing a child with a central disorder, on the basis of subjective auditory responses, as having a 90-decibel hearing loss, is to my way of thinking absurd. This is like taking blood pressure with a clinical

thermometer; one might get some sort of a reading, but it would scarcely be descriptive of a dynamic state of affairs.

DR. IRA J. HIRSH: On this second question, concerning the use of tests for identifying a pathology, Madam Trenque writes—The first question to be answered is, is the lesion situated in the middle ear or in the internal ear? In her first paragraph she points out the well-known observation that when hearing loss exceeds approximately 60 decibels, one can assume that the inner ear is involved. Now, if the deafness is less than 60 decibels, she has suggested that one make a bone conduction examination using conditioning techniques. She suggests also that if the child is not well enough developed to accomplish the test procedure, one can use vocal tests to attempt to resolve the problem. The child who has a transmission deafness, or conductive deafness, will repeat correctly familiar words that he knows, if one raises the intensity sufficiently above the threshold. A child with trouble in the internal ear, even if one raises the intensity far above the threshold, will distort certain words, and the intelligibility will be below 100 per cent.

The manner in which the child has developed language and speech is equally a good indication. If the deafness is severe he will speak very little if at all, and the words that he uses will be simple and childish. If the deafness is less severe, the child will not acquire complicated words. The syntax will be defective and certain consonants will be deformed. The child with a conductive deafness, with onset before the age of acquisition of speech, has a poor vocabulary but good articulation. He presents only a more or less severe language retardation, and in effect he is troubled by a kind of all-or-none phenomenon: either the sound is too weak and he doesn't hear it, or it is strong enough and he perceives it without distortion.

As far as indication on the precise etiology of deafness is concerned, in cases of middle ear deafness, these factors are given by other clinical observations than the auditory tests; for example, malformations of the ear, otitis and toxicosis. In case of deafness of the internal ear most of the time the etiology is difficult to establish in a definite manner; difficult

to establish with certainty. The nature of the auditory residuals and the form of the audiometric curve do not actually permit us to identify the etiology. It is possible that the examination of a large group of children where the diagnostic etiology is certain will produce these interesting results in the future.

From her own observations, she notes the following: that meningitis is followed by rather severe destruction, with almost no hearing residuals or perhaps only islands in the low frequencies. Rubella, on the other hand, gives curves situated between 60 and 80 db. on the audiogram, and more or less horizontal. Hereditary deafness appears to present a polymorphism in audiometric curves. Nothing is identified with anything, I presume, in hereditary deafness. Within the same family one finds a subject with a falling curve; another with a horizontal curve; in a third, complete deafness.

The behavior of the child during the auditory tests very often permits the observation of phenomena superimposed, which point toward a central location for the deafness. Such troubles can be motor, mental, psychological. The test with a peep show and with pictures, from her point of view, are particularly interesting. She gives, third, indications about the development of the deafness. Auditory tests can permit us to follow the evolution of the deafness. We have thus observed, for example, a functional recuperation of the order of ten or twenty decibels in children submitted to auditory training. That is to say, the curve was raised, the threshold was lowered, during the first ten months of training, then it stabilized. She has observed also a remarkable stability in the audiometric curves. Deaf children whom they have followed are in a large majority those who were believed at first, from otological practice, to have hearing which does not continue to deteriorate.

In conclusion, on this second question, the results of hearing tests, oriented toward auditory pathology, are limited, and it is absolutely necessary to supplement these tests in all young children by examining their mental capacity, their language and motor development, and their psychological factors.

DR. ROBERT GOLDSTEIN: Studies of pathologic anatomy provide us with only limited evidence that results on various hearing tests can be associated with various pathologic conditions. This is mostly because studies relating hearing losses to histopathology have been restricted largely to peripheral auditory structures, and then mostly in adults. In the studies of pathologic conditions in the central nervous system the accompanying audiologic studies have, in general, been inadequate. We can say, however, that loss of auditory sensitivity is associated with organic defects of the auditory nerve and cochlea; problems of language comprehension and production with organic defects of some portion of the central nervous system. We have very little knowledge of how much or what kind of central nervous system lesion may be responsible for loss of auditory sensitivity, or of how much damage there is in the periphery in persons with language dysfunction.

We have available considerable information about some children, which allows us to compare results of hearing tests with educational progress, with development of skills of communication, and with general behavior. In some of these children the pathologic basis of their auditory disorders are quite certain from other neurologic or physical defects, and from medical and developmental histories. In the absence of any clearly defined pathologic condition, we can still infer the pathology on the basis of similarities in results on hearing tests and in educational development to children in whom the pathologic bases of their disorders are more certain; and yet, with all this information we cannot say with certainty that the results of hearing tests, or of educational progress, tell us more than: *1.* there is a reduction in sensitivity to sound, probably from a defect of the peripheral auditory mechanism; *2.* there is a disturbance of auditory perception or of linguistic development probably because of some organic abnormality of the central nervous system, or C.N.S.

When we examine very young children we do not have the opportunity to use hindsight from educational development to aid us in determining the nature of their auditory disorders. We may have other than hearing tests to help us with our

assessment, but let us assume that we must make a judgment on the basis of hearing tests only. What kind of information can we get from them to help us decide whether the organic abnormality is in the periphery, in the C.N.S., or in both? In the limited time available, let us restrict ourselves to generalities, acknowledging in advance that there are execptions to all of the generalities.

The child with a peripheral defect will usually startle fairly consistently to sounds above his threshold. The child with a C.N.S. defect may react at first to very weak stimuli, but soon becomes refractory to very intense sounds; or he may not react at all, even to the most intense sounds.

If thresholds of auditory sensitivity can be reliably determined, children with peripheral lesions most often will show sloping audiograms with the high frequencies most severely affected. Children with abnormalities of the C.N.S. or central nervous system, may have normal thresholds, or frequently, hearing losses of about 60 db for all frequencies.

Within any one test the child with a simple peripheral defect will usually show consistency of responses within the limits imposed by his age. The child with an abnormality of the C.N.S. will usually not be consistent in his responses; that is, his apparent threshold may fluctuate unpredictably by as much as 30 to 40 db. Often it is impossible even to estimate the threshold for this child.

A child with a simple peripheral defect is more readily conditioned than a child with a C.N.S. defect to respond voluntarily to auditory signals. This difficulty may be carried over into the conditioning of electrodermal responses to sound.

In audiometry employing the electroencephalograph, the child with a C.N.S. abnormality may sometimes show normal auditory sensitivity through electroencephalic responses while asleep, even though while awake he does not respond consistently to very intense sounds in other tests. The child with a peripheral defect will show the same thresholds through electroencephalic responses when he is asleep as he does through overt responses when he is awake.

When there are both peripheral and central abnormalities

in the same child the consistency or inconsistency with which a child responds may be the most significant differentiating criterion. In such instances, however, we must derive additional differentiating information from other tests and observations.

DR. SILVERMAN: We will refer now to the third question, and that is, what particular testing techniques have you found to be most successful in yielding answers to either or both of the first two questions?

DR. WILLIAM G. HARDY: As to the third question, a good part of this has already been discussed. Dr. Goldstein just added more by implication. I should like to make a few more observations.

I believe we have all learned over the course of years that a fundamental technique is to get the clearest kind of evidence we can from parents who are thoughtful, careful observers. Oftentimes we find that when we work with parents who have been thoughtful and clear in their observations, much of the clinical fancy-work that we do is simply corroborative.

In the course of time we have come to value extremely highly the parents' answer to a simple question that has to do with the child's development; it produces particularly good dividends when there are other small children in the family. "When this baby was three or four months old—not more than that—did you have any question about his hearing?" Oftentimes, when the ultimate picture indicates an aphasoid disorder, they answer "No." Oftentimes, when there is a severe central aberration of one sort or another, the answer is clearly "Yes." Oftentimes, the parents had not observed anything untoward, but this negative fact, itself, is important. What we want to know, of course, is what can appropriately be called an auditory orienting reflex which should be present in infants who both receive and perceive sound.

We like to see what children do in a controlled sound field, under highly structural circumstances, and watched by at least two trained observers. Many kinds and levels of noise

and sound are employed, the general type and range of which is clearly known. Now, a three-month-old child does not wave his arms around and point to the sound-source, but one can see all sorts of reflexes and reactions, often localization; sometimes an interruption of his activities. This last is a kind of negative response which, I believe, is entirely valid when judged as a reaction. Usually, when we see these responses, and help the parents to recognize them, we return to some of the details of parental observation and jog their memories a bit. Thereafter, they come through with many more comments of their own about things they have seen the child do, but have not believed their own eyes, because somebody had told them that their child was deaf. This, to most parents, means a total thing (rare, indeed), so they stop expecting the child to respond to sound.

I feel that I must take sharp issue with Dr. Trenque, *in absentia*, on the etiology and the presumed fact of total deafness, because, from her description I do not know what she means by deafness. We see hundreds and hundreds of post-meningitic children over the years, and I do not remember one that was totally deaf in the sense of complete loss of auditory reflexes. For the most part, I believe, this business of the total, bilateral destruction of the peripheral mechanism is a thing that belongs in history. Medicine in the past 20 years has changed all this considerably. We are all well aware of the total involvement of unilateral impairments from various causes, but bilateral destruction is something different. Of course, in an isolated instance, meningitis can still result in this but, fortunately, in communities with good medical care a child does not go two weeks without hospitalization and diagnosis. If he did, one suspects, he would probably emerge totally deaf now, as he used to.

I shall not say anything more about galvanic audiometry. Our views on that are well known in the literature. We are beginning to work seriously on E.E.G. audiometry, which has great possibilities, not only in measurement of function but also in furthering information about specific sites and modes of neuro-physiologic dysfunction. Let me return to a comment made before. If one is dealing with a child who has

some language and speech, the real point is a thorough test of speech-hearing. This, too, needs to be believed. A child who gives a subjective pure-tone audiogram of approximately 80 db in the speech-hearing range, but who then demonstrates a speech-hearing threshold as good as 40 decibels, is not an educatively deaf child. Another small note of warning: we see many children in the course of a largely consultative service who apparently have central auditory disorders, but who have been forced to use the heaviest duty hearing aids with maximum gains. They have not wanted these aids, for the obvious reason that they have been given too much sound. This warning will bear reiteration, for it is something about which great care must be taken in the field. At stake here, in part, is a wide-eyed acceptance of a concept of deafness as a total kind of behavior, as though one were talking about a specific entity, always related to the same tissue, the same tissue-damage, and the same dysfunction. It is extremely important in dealing with young children to determine whether the problem is peripheral or central, or combined—whether it involves both hearing and listening, or something else entirely; we also find—and believe that this is sensible practice—that oftentimes when one is quite sure that the peripheral mechanism functions fairly well and that there is evidence of a central involvement, a very mild-gain hearing aid may be salutary, largely as an attention-centering device. This is a matter of judgment and experience, not only in terms of a test-picture but also all the rest of the picture which, to us, involves a most thorough otologic, pediatric, neurologic and often psychiatric appraisal.

We were asked to discuss hearing tests. One idea that has emerged from this discussion is that everybody who has spoken on these topics has agreed that hearing tests alone are not sufficient to describe these problems; more than that, that any one hearing test is totally inadequate to describe even a major part of the functions that relate to what I have been calling "reflexive hearing."

Just two more points: I should like to commend attention to Dr. Derbyshire's use of the term *aphasoid*. This is a good term, because it does not have a direct relationship with the

classical neurologic history of the *aphasia* that many of us have lived with. More than that, it stresses the idea that we are not dealing with a little category of specific damage that can fit into a cubby-hole; the "oid" simply means *like*. When a child is described as aphasoid in behavior, the describer is on safe ground, because he is presumably saying that the main trouble is not auditory, and that he wishes he knew better how to describe what is wrong with him.

This world of ours needs to wake up to the needs of children who have these diverse, diffuse central language disorders, with or without central or peripheral auditory disorders. Some of you gentlemen here at the Central Institute for the Deaf may forget that you are sitting in the "angel's chair." You have a curriculum and a faculty capable of moving a child back and forth, at need, from one stream of training to another. To my knowledge, there are precious few other places in existence where this second problem, this aphasoid behavior, is even recognized, let alone where somebody is trying to do something about it.

DR. IRA J. HIRSH: The usefulness of the test depends more on the method employed than on the particular test technique. Auditory tests alone are incapable of resolving the problem as the risks of error are too great. Auditory tests must be included in an ensemble of tests. We ourselves proceed in the following manner: in the first place, we make a very complete interrogation of the parents on the history of the child's genetic development; his motor behavior, his emotional, sensory, and intellectual behavior; the development of his language, and his family. In the second place, we study the mental age of the child. There exists in France a battery of non-verbal tests, established by Madame Borel-Maisonny, that we consider sufficient for a primary discrimination. What we wish to know here is whether or not the child has a substantial mental retardation, and whether he is capable of accomplishing the audiometric tests. In the third place, we study his language. This language evaluation has two objectives: first, to analyze difficulties in the language behavior itself. The alterations in language can be subsumed under two groups of phenomena. First, the tonal quality of

the voice, the rhythm and articulation; when these are very altered the child's language is only a babble, (French, charabia), only once in a while modulated by a recognizable word. The second concerns the understanding of words and their memorization, changes in syntax and automatic responses and verbalizations. The first category is the fact of peripheral deafness, and the second indicates disorders of central origin.

Fourth, we proceed to the auditory tests. We always use tests with noise, or pure tones, and the spoken voice. The principal basis for the voice tests, to which we attach great importance, is the utilization of responses that the child already has, and these can be established in collaboration with his parents. Can the child respond to his name; some words that he seems to understand; what are some of his vocalizations, the words that he can pronounce and others that he can understand? The tonal test that we find most useful is the peep show, because it can be used relatively early, in certain intelligent children at the age of three years, in others at four years of mental age. This age is precisely the one at which we wish to begin intensive training. The second reason for using the peep show is that it is very precise. Our experience in this area is abundant. We have been using it for seven years. Third, the behavior of the child during the course of the test is rich in revelations of his own character and capacities for attention, self-control, comprehension and emotional behavior.

Among the vocal tests our preference is for a test with pictures. We have assembled a large collection of pictures, among which we always find objects or animals that are interesting to a child. We name them with a term that he uses himself, or that he can learn. If a child does not speak, but is capable of designating these objects with some name or other, even though incorrect, this name is used in the test. The test permits us to learn very rapidly the capacities both for hearing and for lipreading in the child.

DR. ROBERT GOLDSTEIN: For every child we try to get as much information as possible from extensive case histories, from psychometric examinations, from neurologic examina-

tions when possible, and from careful observations of general behavior. For today, however, I shall limit myself to the usual kinds of hearing tests we use to learn the nature of auditory disorders, in order to make educational recommendations.

In all of our hearing tests we look for at least two things: one, the weakest sound to which a child will respond, that is, his threshold; two, the manner in which he responds to sound.

First of all we look for overt reactions to sound, either startle responses or voluntary responses. Ordinarily, it is difficult to obtain startle responses at a child's threshold, but if we begin with weak sounds first and gradually increase the intensity, we may get the first reaction close to threshold. Almost any child will cease to react to the same kind of sound, even at high intensities, if that sound is repeated frequently. Variation in the kind of stimuli does much to delay this adaptation to sound. The child with a C.N.S. abnormality tends to ignore sounds sooner than the child with a simple peripheral abnormality.

Whenever it is possible, we attempt to condition a child to make a voluntary response, such as removing a peg from a board when he hears a sound. The child with an abnormality of the C.N.S. is much more difficult to condition than a child with a peripheral defect. In any case, we extend ourselves as much as possible to condition a child in order to get him to respond at the limit of his sensitivity.

When our findings on the basis of overt behavior are too vague to be dependable, we try to determine a child's auditory threshold indirectly by using electrophysiologic reactions to sound as indicators of "hearing." We attempt to condition electrodermal responses, better known to you as GSR, to sound with electric shock as the unconditioned stimulus. Or we examine electroencephalic responses to sound, that is, changes in the brain waves in response to sound while the child is asleep. We do not attempt to condition electroencephalic responses. In both electrodermal and electroencephalic audiometry we try to be as objective as possible in our judgments of electrical responses, which we analyze, by the way,

only after our test is finished; however, we do make note of any overt responses during our tests.

As in the case of conditioning voluntary responses, it is generally more difficult to condition electrodermal responses to sound in children with C.N.S. defects than in children with peripheral defects. This is by no means a clear differentiating criterion because there are many deaf children and many normal children who are also difficult to condition.

In our brief experience with electroencephalic audiometry we have found that it works as successfully with the children we call aphasic as it does with deaf children. We see on a few occasions that children with C.N.S. abnormalities give responses to significantly weaker sounds than ever consistently elicit voluntary or electrodermal responses. Ordinarily, though, there is good agreement among all tests as to threshold of auditory sensitivity when there are reasonably consistent responses in each of the tests.

Sometimes we are successful in determining auditory thresholds by these indirect techniques, and sometimes we are not. Most often, however, we get enough information to confirm, at least partially, what we are able to observe in the overt responses.

In general summary, we try to condition voluntary responses to sound whenever possible and rely on startle responses when we cannot condition a child. We push to the limit of auditory sensitivity but we note responses to suprathreshold stimuli as well, as indication of a child's ordinary reaction to sounds. We attempt indirect determination of thresholds through electro-physiologic responses to sound when we cannot obtain reasonably consistent overt responses. The child with loss of auditory sensitivity because of a defect of the auditory nerve or cochlea, that is, the deaf child, usually conditions fairly easily within the limits of his age, is reasonably consistent in his responses, and usually shows a greater loss of sensitivity for high frequencies than for low frequencies. The child with a defect in auditory perception and in linguistic development, because of some C.N.S. abnormality, is more difficult to condition, is usually inconsistent

in his responses, and most often shows no loss or only a moderate loss of sensitivity.

DR. SILVERMAN: Dr. Myklebust will give his answers to the questions in a single presentation.

DR. HELMER MYKLEBUST: Emphasis on the ways in which hearing tests can be useful in making educational recommendations is highly desirable. Often hearing tests have been analyzed in terms of their significance in psychophysiology without reference to the implications of the results for the

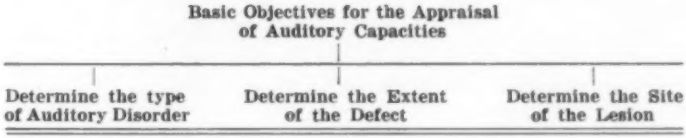

Basic Objectives for the Appraisal of Auditory Capacities
Determine the type of Auditory Disorder
Determine the Extent of the Defect
Determine the Site of the Lesion

Fig. 1.

The Primary Types of Auditory Disorders.

Peripheral Deafness	(End-Organ)
Central Deafness	(Auditory Pathways)
Psychic Deafness	(Mental Illness)
Perceptual Disorder	(Listening Defect)
Receptive Aphasia	(Language Disorder)
Auditory Agnosia	(No Sound Meaning)
Overlapping Conditions	(Multiple Disorders)

Fig. 2.

child's education and general adjustment. When we study the objectives for appraising a child's auditory capacities we find there are at least three basic goals: (a) to determine the type or nature of the auditory incapacity, (b) to determine the extent or the degree of the defect, and (c) to ascertain the site or sites of the lesion. This is shown schematically in Fig. 1.

There are six conditions encountered in the appraisal of auditory disorders in young children, as shown in Fig. 2. The seventh possibility is that more than one deficiency is present, necessitating the determination of overlapping conditions.

Peripheral deafness refers to end-organ defect. Central deafness means that the auditory pathways in the central nervous system have been impaired and that the nerve impulses do not reach the interpretation area in the auditory cortex. This impairment falls essentially between the cochlear nuclei and the medial geniculate bodies.

Psychic deafness is the condition whereby the child has adequate capacities organically but rejects sound for reasons of emotional disintegration. It is our experience that psychic deafness in young children occurs mainly in psychotic children, but might be present in the severely neurotic.

Receptive aphasia occurs as a result of damage to the interpretation area in the temporal lobe. It is a symbolic language disorder and is present with considerable frequency in young children.

Auditory agnosia refers to the condition resulting from gross damage to the auditory cortex. Apparently this condition occurs only rarely. When it is present the child cannot attach meaning to any sound although he has normal hearing acuity.

Auditory perceptual disorders are complex and may affect the normal auditory process in various ways. Figure-ground may be disturbed with the result that the child cannot use the auditory field selectively; thus, he can hear, but he cannot listen. Auditory memory and discrimination also may be involved.

Overlapping conditions refer to the presence of more than one defect. Perhaps one of the primary problems relative to hearing impaired children is the multiple handicap of hearing loss and brain damage.

Complete auditory appraisal assumes that each of these conditions has been considered and that the specific type of auditory disorder has been determined.

Now we come to the second major objective, the determination of the extent or the degree of the defect. If peripheral deafness is present it is essential to ascertain the degree of the hearing loss. For educational purposes the most sig-

nificant single factor in this connection is the extent to which the deafness has affected language acquisition and usage. Four major classifications of degree of deafness relative to educational recommendations are shown in Fig. 3.

Children with a moderate hearing loss do not require specialized educational procedures. Their language abilities fall within normal limits. Those who are severely hard of hearing require special educational methods, but they benefit substantially from the use of amplification. The profoundly

The Degree of Deafness.
Hard of Hearing (Moderate).
Heard of Hearing (Severe).
Profound Deafness.
Deafened.

Fig. 3.

The Site of the Lesion in Auditory Disorders.	
External Ear	(Atresias)
Middle Ear	(Conductive)
Inner Ear	(Nerve-cochlear)
Auditory Pathways	(C.N.S.)
Auditory Cortex	(Temporal Lobe)

Fig. 4.

deaf child seems to gain very little educationally from amplification, and thereby is dependent upon the specialized techniques developed specifically by educators of the deaf. The deafened child is one who has sustained profound deafness after five years of age and thus retains his language abilities; when profound deafness is sustained prior to five years of age usually the language which had been acquired is not retained.

The third primary objective of the auditory appraisal is to determine the site of the lesion. Five major sites of lesions to be considered in appraising auditory capacities are shown in Fig. 4.

Lesions of the external, middle, and inner ear traditionally are determined by tests of acuity, using the bone and air

conduction methods. The most common type of deafness in young children is that which results from deficiency in the inner ear. Frequently, inner ear lesions can be predicted from the degree of the hearing loss; when losses of 60, 70, 80 db, or more are present it is apparent that the loss does not derive from a simple conductive involvement. Lesions of the central auditory pathways and of the auditory cortex are more difficult to determine objectively; however, some test suggestions are given later.

Age of Onset of the Auditory Disorder.

Prenatal Period	(Before Birth)
Pre-speech Age	(Birth to 2 yrs.)
Early Language Period	(2 to 5 yrs.)
Late Language Period	(After 5 yrs.)

Fig. 5.

Etiological Classification of Peripheral Deafness.

Endogenous—(All hereditary)	22.6%
Exogenous—(All non-hereditary)	
(a) Meningitis	11.3%
(b) Other diseases	27.8%
Undetermined (Unknown)	38.3%
N = 822	100. %

Fig. 6.

Two other significant determinations from the point of view of educational recommendations are the age of onset and etiology. These do not derive directly from the auditory test itself, but they are factors that must be considered when interpreting the results. The importance of age of onset is indicated by the different nature of the educational recommendations when deafness is sustained in the pre-speech age, or after five years of age. The importance of etiology is indicated by the higher incidence of multiple handicaps, such as deafness and brain injury, when the deafness results from rubella, meningitis, or certain other diseases. The primary classifications of age of onset in young children are shown in Fig. 5.

The primary etiological classifications are shown in Fig. 6.

That etiological factors are significant in terms of educational recommendations is apparent from the fact that research reveals significant differences between the exogenous and endogenous in such areas as intelligence, motor capacities, and educational achievement.

No one test of auditory capacity can satisfy all of our demands or goals. Actually three essentially different types of tests are required. This is illustrated in Fig. 7.

Diagnostic Appraisal of Auditory Capacities.

Peripheral Nervous System—End-Organ	Central Nervous System C.N.S. Pathways and Auditory Cortex	Psychological Integrity Mental and Emotional Processes
Auditory Acuity: ↔	Integration and Use: ↔	Psychic Auditory Behavior
(a) Conditioned play audiometry.	(a) Listening Tests.	(a) Scanning Tests.
(b) Sound field, speech, gross sounds and pure tones.	(b) Localization Tests.	(b) Foreground-background.
(c) PGSR.	(c) Perceptual Tests.	(c) Vocal monitoring.
(d) EEG.	(d) Language Tests.	(d) Imitation and Echolalia.
(e) Behavioral Symptoms.	(e) Pain Sensitivity Tests.	(e) Comprehensive-Selective.
	(f) Behavioral Symptoms.	(f) Behavioral Symptoms.

Fig. 7.

Most tests provide relevant information concerning the integrity of all three of these avenues. For example, if a child is difficult to condition, either with PGSR or in conditioned play audiometry, there is a presumption that even though he might have a hearing loss, he is also brain damaged, mentally ill, or mentally deficient; however, some tests are more useful than others for ascertaining a specific type of integrity. We have found listening tests to be highly useful in the study of children presenting problems of the brain damage type. In general, receptive aphasic children have auditory perceptual disorders, and thus cannot give the sustained attention required in listening. We are in the process of standardizing a battery of auditory perceptual tests on normal children and those with auditory disorders,

and these tests show promise for use in diagnosis. The implications of this study are shown in Fig. 8.

Normally we select sounds from the auditory field according to organismic exigencies at any particular moment. This is illustrated by the arrows showing normal shifting of figure-ground. We see also that a basic function of background hearing is scanning of the environment in order that the organismic homeostatic relationships can be maintained. In contrast, foreground hearing serves the basic purposes requiring listening, especially for the purpose of communication.

Auditory Perceptual Functioning and Figure-Ground Behavior.

Background Hearing	Normal shifting of Figure-ground	Foreground Hearing
Mandatory scanning of environment		Listening
Signaling-warning		Receptive language
Homeostatic awareness		Immediate needs of the organism

Fig. 8.

In general, study of young children having auditory disorders suggests that they fall into groups on the basis of their auditory perceptual behavior. Those having impaired hearing lose the background function of hearing and, therefore, are restricted to foreground hearing. In psychic deafness the child relinquishes inter-personal relations by rejecting foreground hearing, but he retains the more peripheral or primitive background use of hearing. The brain damaged child cannot structure the auditory world, so he is a victim of the entire sound field. He cannot select an appropriate sound and attend to it to the extent that the organismic needs are achieved satisfactorily. In a recent publication Dr. Neff reported similar behavior in cats after ablation of parts of the auditory cortex.

The child with central deafness seems deficient on tests of localization; this is in contrast to those with peripheral deafness; furthermore, these children seem to be reduced in pain level responsiveness. They might have normal acuity by PGSR but do not have ability to monitor their vocalizations.

A number of other characteristic responses might be given.

Psychic deafness in young children frequently can be determined most satisfactorily by informal tests, or by a combination of formal and informal techniques. Because these children tend to reject foreground hearing the test which is limited to background responses is most suitable. Conditioning techniques frequently are unreliable with this group. Tests requiring scanning of background, monitoring of vocalization, and selective comprehension have been helpful in ascertaining whether such children have normal hearing and are rejecting sound for emotional reasons.

In young children presenting complex auditory disorders the hearing test alone frequently does not make it possible to make a differential diagnosis. Often psychological, psychiatric, and neurological study must be included. The total maturational aspects and the behavioral symptomatologies are critical factors.

Of a group of 325 pre-school age children with auditory disorders there were eighty with peripheral deafness, sixty-eight with deafness with central involvement, one hundred and forty-two having aphasic and other types of central involvement, twenty-four with psychic deafness, and eleven mentally deficient. A revealing aspect of this study was the high incidence of children having both deafness and brain injury and the slightly higher incidence of children having auditory disorders other than peripheral deafness. This seems to us to highlight the importance of differential diagnosis and the implications for educational recommendations.

DR. SILVERMAN: What we shall do first is to ask the panel to react with each other.

DR. HARDY: Considering the independence of this group, I think there is a tremendous unanimity involved. I do not know where Dr. Hirsh stands. I am becoming increasingly concerned about the use of the term "brain-injured" as a genetic reference to so many of these problems. We see many more children who get that way from pre-natal events of one sort or another wherein there is no exterior evidence of injuries. Neurologists examine these children and keep

telling us that they can see no other evidence of brain injury, and our psychologists keep saying they can see no other particular evidence determinable except auditory perception.

You have to be very careful about this concept so that it does not become a label attached to many of these children; because, in all truth, ninety-nine per cent of our school people will not accept a "brain-injured" child, whereas a deaf one is all right. I would like to know what the panel thinks about that.

DR. MYKLEBUST: Well, I might say that I quite agree that we need a better term; also, that, as we use the term, we do mean central involvement, not necessarily literally an involvement of the damage type. It is entirely possible that some of these are biochemical, of course.

I would like to comment on the latter part of Dr. Hardy's comment just to this extent, that I suppose, central involvement, or damage, whichever kind of term we are going to use, is in the state today where there is no one key to it. Sometimes the EDG shows it; sometimes a neurological shows it; sometimes the psychological shows it, etc. Certainly, motor tests and other tests of this type are frequently useful in bringing this into the foreground. It is essential that we use a multiphasic approach of study to this condition at this time.

DR. GOLDSTEIN: I would like to make two comments about the use of the word or term "brain-injury"; one is that I am in partial agreement with Dr. Hardy that a person can have organic injury to the brain and have no obvious problems in communication. He may have perfectly normal hearing, speech and language. A person who does have problems in communication that cannot be attributed to a loss of sensitivity, to mental deficiency, or to psychic disturbance, may very well have a language disturbance of some sort presumably due to some abnormality of the organic central nervous system; in fact, we have enough indications to accept the aphazoid or aphasic behavior as a symptom of some central nervous system abnormality.

I have a greater objection to the word "brain-injury" in

parallel with the term "deafness." I think we should say either "ear-injury," "brain-injury" or "deafness" and some other language disturbances.

DR. HIRSH: Dr. Trenque has written a summary page which she entitles "Reflections on the Problem of Auditory Testing of the Young Child, and the Data that such Tests Supply."

At this stage of our experience, we consider that there are two categories of children: relatively easy ones and difficult ones. The relatively easy ones are the following: First, purely deaf children, that is, children who are only deaf without other deficiencies superimposed. One knows now the behavior of purely deaf children; their characteristic difficulties, and the retardation that follows the deafness in intellectual performance. The diagnosis is fairly easy, and the measure of the amount of deafness can be established with precision after the age of four years. The second category of relatively easy cases are mental defectives. These are equally easy to screen. It is more difficult to say whether a partial deafness is supplemented by mental retardation.

The difficult cases are composed of the following groups: children who have important psychic difficulties; aphasic children; deaf children with an intellectual or psychic deficit. In our opinion, only a prolonged observation will permit the establishment of a diagnosis or prognosis that is precise. Compensation for deafness depends essentially upon mental capacities and the social adaptation of the child. One can only hope, even after a complete test of hearing, intellect and psychology of the child. The real capacities for overcoming deafness cannot be appreciated definitively even in the course of the first stage of education, that is, at the acquisition of concrete vocabulary, reading and writing, but only after the child has acquired more complex notions of time, cause, attributes, etc.

DR. MYKLEBUST: I think there is perhaps more agreement on this question than we had anticipated. I suppose I could raise the question now as to whether we can classify children

having certain kinds of language disorders as aphasic. I find it difficult to see why we should not do so if the condition is comparable to that traditionally referred to with adults. I do not see why we should confuse age of onset with classification. The fact that the child sustains this prenatally or at the time of birth does not alter the basic classification.

In deafness we specify early deafness by congenital deafness; in psychiatry, they use the term schizophrenia. Childhood aphasia. I wonder what you think about that.

DR. GOLDSTEIN: I would like to speak in support of that comment. I think that simply because the term "aphasia" grew up around adults does not make it the particular property of adults. People have objected to the use of "aphasia" in children because of the obvious differences in behavior of the adult aphasic. They say that the person who has sustained an accident is so different from the child who never developed language, but, have you ever stopped to compare an adult who suddenly loses hearing to a child who never had any hearing. There is a tremendous difference in many aspects between these two people, and yet, if they have certain symptoms in common, we have no objection to calling the adult "deaf" and the child "deaf."

DR. HARDY: I object to calling either of them deaf. Let us consider how much hearing they have.

I would like to play along with this idea of childhood aphasia with a proviso or two: first, the field of neurology is beginning to learn what we are talking about in terms of functional and developmental aspects of these problems. I am not sure that is a general truth at present; second, everybody who addresses this concept of childhood aphasia really makes a serious effort to include and distinguish this business of auditory perception or imperception and stop talking about, let's say, a peripheral child with aphasia. The only qualification is that these things be made inclusive and exclusive in one way, whether one is discussing auditory information as the lack of development, or damage to the centers which have to handle that information as the lack of development, and how these two get together.

We should give our audience an opportunity to direct questions to any member of the panel.

DR. HIRSH: I apologize for not being able to defend some of Dr. Trenque's points here. There were certain paragraphs in which I panicked before I got to the end, and this will be published in full when we publish the proceedings. I thank Dr. Bocca, who brought Dr. Trenque's paper here. He has seen her recently, and perhaps, from the discussion that comes from the floor he may wish to make a comment or two as to his reflections of her point of view on these matters.

DR. BOCCA: I have no comment.

DR. SILVERMAN: We shall now give our audience an opportunity to direct questions to any member of the panel.

DR. BRUCE M. SIEGENTHALER: I would like to direct a question to Dr. Myklebust. In your last slide you showed the results of the classification of three hundred and some children. Will you comment as to what general population that has been drawn from? What are the factors that led to picking out these particular three hundred and twenty-five children?

DR. MYKLEBUST: That is a very good point. I am glad it comes up because I meant to mention, that this is a selected sample. This is a sample of children who come to a hearing diagnostic center in a medical school. They are definitely selective factors. They have come there because they have very real problems.

DR. SILVERMAN: May I ask a question: do you think that because we become interested in a certain kind of child, we may be looking at a very obscure sample in the way we operate?

DR. MYKLEBUST: I would agree with that entirely; and we must be careful about selected samples. That is why, in another survey which does include children throughout the nation, we have stratified samples from all types of schools and age levels and so on. I think it is a very important question. I think it is very important to mention that the data there necessarily represent the basic things.

DR. R. N. REILLY: During the discussion of the successful techniques none of the members of the panel have mentioned the question of frequency modulation in testing young children. When I was in the Netherlands three years ago, Dr. Huizing was using frequency modulation in testing babies and young children, and during the past six months I have used it with quite a good deal of success. I wonder what the comment of the panel would be in relation to that?

DR. HARDY: I think, Dr. Reilly, that this simply reflects the time. We have used frequency modulation for six or seven years as part and parcel of the pure tone presentation in subjects. It is a wonderful attention centering device and oftentimes, as you play back and forth between a steady pulse tone and then introduce modulation, you get responses you cannot get otherwise, particularly with C.N.S. involved children, I think.

DR. MAURICE H. MILLER: Dr. Myklebust mentioned that the child with a central disorder will have difficulty, or may have difficulty, in localizing sound. Will not a child with a peripheral problem also have difficulty in localization if there is a large difference in the acuity between ears; and is this not a factor of the difference between ears rather than with the site of the lesion?

DR. SILVERMAN: May I repeat the question. The question is first in the form of a comment that Dr. Myklebust indicated that difficulty in localization might indicate a lesion of the central nervous system. Dr. Miller raises the question as to whether that might not be true of end-organ or middle ear deafness, for that matter, and may be related to a difference in sensitivity in the two ears.

DR. MYKLEBUST: Yes, of course. Difference of degree of involvement of the two ears might very well be affecting localization response. As such, we can do nothing but accept it and entirely agree that it is a very real possibility.

I suppose I am referring to something more like this; that in certain children, and this is not specifically those with, let's say, a simple peripheral involvement; not only do they not localize, but they do not attempt to localize. This is

perhaps not so true of the individual with the difference in the two ears.

It seems to me that it is something worth watching and worth testing for increasingly. I think we have a great deal to learn with this localization and its validity and reliability and so on. We do feel that there is a correlation between it and damages that cannot be explained on the basis of peripheral involvement.

DR. HARDY: Dr. Trenque agrees with that observation. I should think that where there is a profound involvement this would not be too critical a point; because, it is our experience that when it is profound, either peripheral or central, it is bilateral; both sides are involved. Oftentimes there are conductive lesions, or acute otitis more severe in one side or the other.

This business of undertaking audiologic appraisal of these young children without a full foreground and background of the best possible work is for the birds, as we may say. You have all the information possible about the organic state of the child. I think that would tend to direct the thinking.

MR. JACK ROSEN: I am particularly pleased to have so much attention called to the central problems, but, at the same time, I would like to call attention to the fact that there is no law which requires that a child have only one problem; even if there were, children would not obey it.

The purpose of this remark is that the central problems are determined upon the basis of symptoms which are not auditory for the most part. It is quite possible, and in fact, very often it does occur that children do have hearing losses and some of these other symptoms at the same time. There is a tendency, when attention is called to a new phenomenon, to disregard some of the older phenomena and techniques; and I would be particularly concerned about the neglect of careful audiological evaluation before deciding that a child has only a central problem or only a psychic deafness, because this can lead to serious problems. I would also be very care-

ful about deciding this on the basis of gross tests such as responses to vocalization, the voice tests which are sometimes correlated with speech tests but are not the same thing; thus, children with severe high frequency deafness are now sometimes being classified as children with central problems, children with psychic deafness, because they readily respond to voice and yet will not learn speech.

DR. SILVERMAN: I shall now attempt to summarize the panel deliberations.

The panel directed its attention to three questions: one having to do with the goals set, particularly with reference to educational recommendations and the testing of the hearing of young children; two, the evidence for the results of hearing tests that can yield information about the pathology or auditory and other disorders; and three, what particular techniques are used.

The panel seemed to be concerned with the differentiation of end-organ from other types of lesions; and at one time seemed to work on the anatomy; another time on the dimensions of behavior; and still another time on the dimensions of ease of conditioning.

They seem to go from one to the other, and they seem to admit that it was difficult to follow the outline. In one case we got the words "conductive" and "language" in the same column, and in another case we get a little bit of anatomy and behavior in another column; but this, again, is a matter of groping for the universals to describe certain kinds of conditions that are observed.

There seemed to be unanimity with what was stated so succinctly by Dr. Trenque, that there are easy and difficult cases. What we are trying to do, apparently, is to supplement what seems to be a good deal of clinical artistry. All of this is involved. We have to develop more objective techniques.

One thing surprises me, however, about the statements of the panel here, or one omission—and I know they are aware of this—and that is the validation of some of the statements

that have been made. For example, from the audience there was a comment about a test's being good. What are the dimensions of goodness in terms of the behavior of the child as he develops?

I also was surprised that there was as much agreement among the people. I looked forward to bringing them together, and I am delighted that we have had this opportunity to have all of them. I express our gratitude.

II. DIAGNOSIS OF HEARING DISORDERS.

After a brief recess, the International Conference on Audiology reconvened in the Auditorium of the Washington University School of Medicine, St. Louis, Missouri; Dr. Ira J. Hirsh, presiding.

DR. HIRSH: We will now continue this afternoon's symposium on the Assessment of Auditory Function. We have discussed so far tests appropriate to young children, and now we propose to approach an area that represents probably the closest connection between the disciplines of otology and audiology.

AUDIOMETRY IN DIAGNOSIS.

RAYMOND CARHART,

Evanston, Ill.

I.

The subject of audiometry in diagnosis has so many ramifications that one cannot hope to discuss it fully in a single paper; moreover, the subject is one to which both research and clinical contributions are continuously being made, so that any review one undertakes today will seem inadequate tomorrow. Realism requires that the present discussion be restricted appropriately.

To this end, only the relationships between measurement of thresholds for pure tones and diagnosis of hearing disorders

are here considered. The importance of speech audiometry, the significance of supra-threshold function in response to pure tones and to noise, and the value of special techniques, such as psychogalvanic audiometry are obvious. Each of these topics is worthy of extensive coverage, but the present discussion is arbitrarily restricted to a review of major principles in interpreting conventional threshold audiograms and to an examination, through examples, of the fact that the formulation of additional principles may be anticipated.

II.

The air conduction audiogram conveys substantial information of diagnostic importance.

For one thing, air conduction curves are sufficiently typical in some types of hearing loss so that the audiometric pattern suggests a possible etiology to the clinician. The most obvious illustration of such a situation is the notched curve showing loss for high frequencies, which leads one to suspect noise-induced hearing loss. Another example is the gradual deterioration in acuity for higher frequencies so characteristic of presbycusis. In contrast, there is the relatively flat loss of about 50 db. which is slightly more pronounced for low frequencies than for high frequencies. Such a curve is likely to result from marked stapes fixation, from certain other conductive lesions, or from labyrinthine hydrops, if it is of the sensori-neural variety. Again, a trough-shaped audiogram which tends to follow an equal-loudness curve, *i.e.*, an equal phon contour, immediately suggest the possibility of a non-organic hearing loss; thus, while threshold pattern alone never allows definitive diagnosis, the details of the air conduction curve often contribute to such diagnosis. It is, therefore, pertinent to continue an interest in the types of curves which categorize varieties of hearing impairment, and to seek further insight into the inter-dependencies between the two.

As a corollary to the foregoing observation, the relationship between the two air conduction curves obtained from a single patient tends also to distinguish varieties of hearing loss. Bilaterally symmetrical impairments are relatively common in some conditions, such as in endogenous congenital losses, and in presbycusis. Moderate dissymmetry of loss is usually

observed in other conditions; for example, in hearing losses due to anoxia at birth, or to otosclerosis. Other pathologies most commonly affect hearing in one ear primarily or exclusively. One illustration is the complete unilateral destruction of auditory function which mumps sometimes cause. Similarly, acoustic neurinomas, temporal bone fractures, and sudden vascular disturbances usually are unilateral and, hence, effect audiometric results in only one ear. Again, cases with labyrinthine hydrops often show depressed hearing in only one ear, although this condition may manifest itself bilaterally, and hearing loss tends to fluctuate markedly. In consequence of tendencies such as those just mentioned, the clinician can frequently utilize his observations on the audiometric balance between ears as a helpful segment of the information he requires in order to reach his diagnostic judgment.

III.

The fact that the relationship between air and bone conduction thresholds assists in distinguishing sensori-neural involvements from lesions in the external and middle ear is, in the minds of many people, one of the most important diagnostic applications of pure-tone audiometry. The principles involved are generally accepted. It is clearly recognized that adequate determination of bone conduction thresholds requires precautions of calibration and of test administration beyond those which are necessary for good air conduction audiometry. When such precautions have been taken and the patient has an organic loss, the general level of the bone conduction curves indicates the degree of sensori-neural involvement (without distinguishing between cochlear and retrocochlear sites). If the air conduction curve lies at the same level, the total loss may be judged as sensori-neural. By contrast, there cannot be a sensori-neural involvement when acuity by bone conduction is normal. Here, any substantial depression of acuity for air-borne stimuli must be due to a lesion in the transmissional system. In other words, such a relationship indicates a conductive loss. The third alternative is the condition where both bone and air conduction curves are depressed, but where the loss by air is significantly greater than

by bone. In such an instance, the depression of the bone curve bespeaks a sensori-neural component of loss, while the further depression in acuity for air-borne sound is evidence of a concomitant conductive, or a transmissional, loss. One is here faced with a composite loss or, as some would still term it, a mixed loss.

IV.

In recent years it has become increasingly apparent that the clinical audiogram contains a host of subtler clues which can contribute to finer diagnostic differentiation. A few of these are currently known, as is illustrated in the subsequent sections of this paper. It is imperative to remember, however, that the clinical audiogram contains additional clues. Some of these await discovery, while the significance of others needs clarification. The generalization which must be stressed at the outset is that clinicians have tended to discount subtler configurational features of the audiograms at their disposal and, hence, have failed to gain full benefit from these test records. True, much research is needed to catalogue all the aids to differential diagnosis which reside in good clinical audiograms. For the moment, however, the important points are that known clues are not being used fully, and that clinicians, on the whole, are not actively curious about the less obvious significance of the pure-tone audiogram.

V.

Particularly good illustrations of subtle audiometric clues are found in the bone conduction curves characterizing different kinds of transmissional deafness. Mechanical lesions tend to cause distinctive distortions in bone conduction audiograms. These distortions are slight, and they are due to changes in the physical constants of the auditory system rather than to modifications of sensori-neural activity. In other words, the effective agitation reaching the organ of Corti from a bone conduction oscillator can be influenced to a measurable degree by the condition of the middle ear and of the external ear.

Fixation of the stapes, for example, induces such mechanical modification in the bone conduction audiogram. Fig. 1 pre-

sents a record which is characteristic of this condition. The patient involved was diagnosed as having clinical otosclerosis without secondary nerve degeneration. For sake of simplicity, the responses for only one ear are shown in the figure. The significant feature of this audiogram, for the moment, is that the depression of the bone curve which one notes at frequencies above 500 cycles, is an artifact in the sense that the normal state of the patient's inner ear is not revealed with full accuracy. Failure to consider this artifact when interpreting the audiogram would lead to the erroneous conclusion that the patient possessed a mild sensori-neural involvement. Conversely, awareness that this general pattern of mechanical distortion in the bone curve is typical of stapes fixation increases the diagnostic value of the audiometric record.

As one would expect, the mechanically produced modification in bone conduction response consequent to stapes fixation varies from patient to patient. At least in most cases, however, these variations are not so great that they obscure the general pattern of shift characterizing stapes fixation. Most typically, the mechanical shift in the bone conduction curve induced by stapes fixation is of the magnitude and of the shape represented by the shaded area in Fig. 1. That is, an otosclerotic patient with normal cochlear function, which would be revealed by a bone conduction audiogram at normal if it were not for the mechanical shift in responsiveness, will most typically yield a notch-like depression of the bone curve which follows the lower boundary of the shaded area in the figure. The audiogram shown in Fig. 1 did not conform perfectly to this course, but the deviation did not obscure the basic pattern characteristically induced by stapes fixation.

Such a notched depression in the bone curve is less obvious to the eye when secondary nerve degeneration is combined with stapes fixation, but it is nonetheless present. A typical audiometric record illustrating this situation is shown in Fig. 2. The position of the bone conduction curve is shifted and given a downward tilt toward the right by the senori-neural loss which is present in this case. If this sensori neural condition were the only impairment present, there would be a smooth progressive drop in acuity with increase in stimulus frequency; however, the smoothness of this pro-

gression is obscured by the mechanical distortion in the bone curve which stapes fixation caused. Stated conversely, the trade-mark of stapes fixation is to be found in this record, if the clinician wishes to seek it. The break which occurs in the bone conduction curve at 2000 c.p.s. is the preliminary clue to mechanical distortion in the curve. Confirmation is

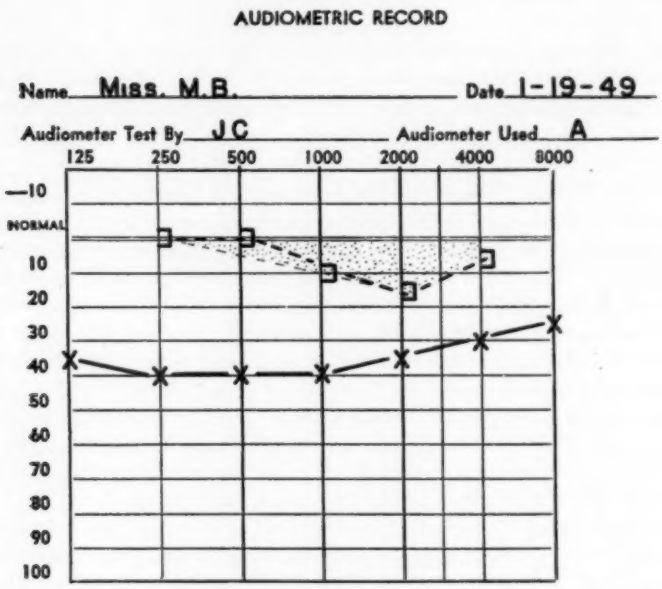


Fig. 1. Hearing levels for the left ear of a 17-year-old girl having clinical otosclerosis without secondary nerve involvement. Stippled area indicates usual magnitude of change in bone thresholds induced mechanically by stapes fixation.

achieved by subtracting the idealized mechanical notch from the bone curve. The residue thus obtained reveals graphically the so-called cochlear reserve, that is, the configuration of sensori-neural acuity which the patient has retained. In the present instance, this residue is characterized by the smooth progression in loss for higher frequencies mentioned previously.

VI.

Many other conductive lesions produce shifts in the bone conduction audiogram. Here, physical similarity among lesions, regardless of etiological classification, will determine a particular audiometric configuration. For example, permanent damages resulting from otitis media can not all show the

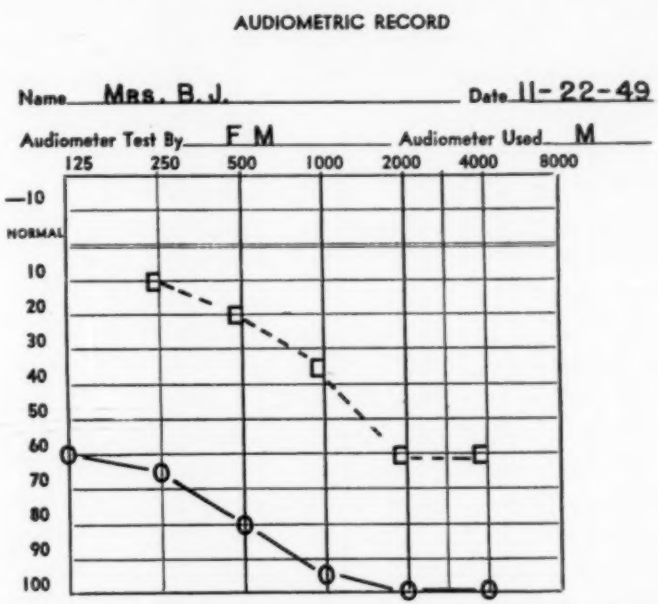


Fig. 2. Hearing levels for the right ear of a 40-year-old woman having complete stapes fixation due to clinical otosclerosis plus secondary nerve degeneration.

same pattern of mechanical artifact in the bone curve simply because they do not all produce equivalent damage in the middle ear mechanism; moreover, until clinicians learn to classify each conductive lesion on the basis of distinctions in the mechanical damage which characterize it, they will be limited in the precision with which they can differentiate among meaningful artifacts in the bone conduction curve.

Certain patterns are nonetheless emerging. It is possible to illustrate in general terms the value which the clinician can gain from further knowledge of the subtle clues that reside in a good bone conduction audiogram.

As one example, consider Fig. 3. Again, the results for only one ear are reported. The diagnosis in this case was

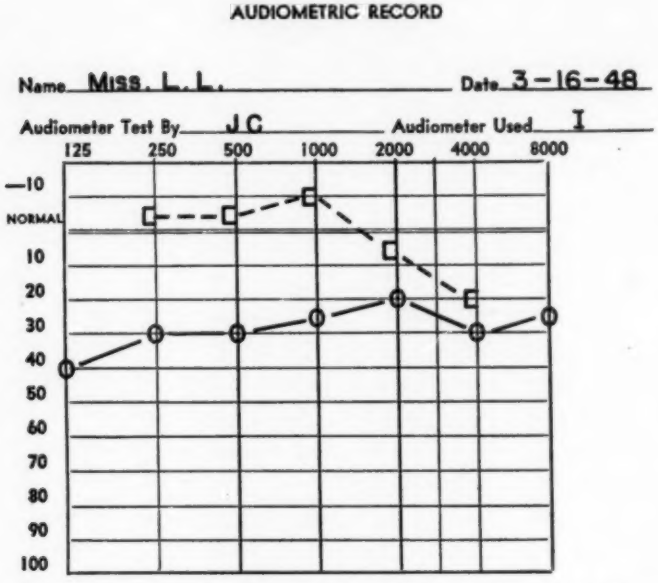


Fig. 3. Hearing levels for the right ear of a 13-year-old girl having chronic suppurative otitis media.

conductive hearing loss due to chronic suppurative otitis media. The important thing is that the responses by bone conduction show a pattern frequently observed when there is disease in the middle ear. This pattern, which was described years ago by Lierle and Reger,[15] is characterized at low frequencies by better than normal responsiveness to bone stimuli, and by reduced responsiveness at high frequencies. In many instances the entire configuration is probably induced me-

chanically. Specifically, the case reported in Fig. 3 probably did not possess either cochlear hyperacuity for low frequencies or sensori-neural deficiency for high frequencies. One reason for arriving at this conclusion is the fact that the patient did not show a high frequency drop by air conduction comparable to the drop by bone. This patient's air conduction audiogram, in other words, has a configuration which bespeaks a conductive lesion without appreciable sensori-neural involvement. There is a second reason for expressing belief that the pattern of bone conduction response under consideration is mechanically induced. Many cases showing this pattern reveal sudden modification in the pattern of the bone curve as soon as the mechanical status of the middle ear is changed. One example which comes to mind is a case wherein the bone conduction curve reverted essentially to normal when the patient's perforated ear drum was closed with a paper patch.

It is likely that clinicians have often mistakenly interpreted minor decreases in bone efficiency at higher frequencies as evidence of mild sensori-neural involvement. Such a sensori-neural involvement is properly assignable only if the air conduction curve incorporates a break in its course that also indicates decrease in acuity for high frequencies. When concomitant depression of the air conduction curve is not found, a minor depression in the bone conduction curve should be presumed to be evidence of mechanical modification in the conductive mechanism. A similar interpretation is assignable to minor elevations in the bone curve.

One must reiterate that it is not currently possible to state the specific nature of the mechanical changes underlying a particular pattern of shift in bone responsiveness. Bone conduction audiometry will have increased diagnostic usefulness when research reveals the relationships which are involved. The first step is to develop sufficient confidence in the existence of such relations so that they are sought out systematically and their meaning is mastered; moreover, the clinician must remember that, if the patient has lesion or pathology in the middle ear or the external ear, the exact picture of cochlear sensitivity is seldom revealed by the bone conduction

audiogram. True, the general level of cochlear sensitivity may be estimated from this audiogram, but the details of apparent responsiveness will often be distorted by mechanical influences. The clinician must make allowances for these influences when estimating the sensori-neural efficiency of a patient with a conductive involvement.

VII.

Much attention has been given in recent years to the concept that anticipable shifts in air conduction acuity are induced by changes in the mechanical impedance of the auditory system. The writings of Metz,[16] Johansen,[10] and Campbell[2] are sufficiently well known so that they need not be reviewed here.

The important idea is that the clinician can find value in these concepts when he is interpreting conductive losses. Here the configuration of thresholds for air-borne stimuli may be viewed as a manifestation of specific changes which have taken place in the physical constants of the ear. Naturally, the clinician is not going to attempt detailed mathematical analyses of individual case records; however, he can profitably bear in mind the general principles which the physicist has made available to him, and which appear consistent with clinical experience; namely, that increased stiffness or decreased mass reduce sensitivity for low frequencies, whereas the reverse conditions reduce response to high frequencies.

Consider, as one example, the case shown in Fig. 4. Only the two air conduction audiograms are here reported. This patient was diagnosed as having clinical otosclerosis without secondary nerve degeneration. The stapes was found to be only partially fixed on the right side, but completely fixed on the left. The hearing loss in the right ear is substantial for low frequencies and mild for high frequencies. According to the principles mentioned above, such a configuration could be either the result of mass decrease or of stiffness increase. Since a substantial decrease in the effective mass of the conductive system is inconsistent with the pathology of otosclerosis, the first of these possibilities may be quickly

dismissed. The alternative, an increase in stiffness, is credible when one remembers that the encroaching spicules of otosclerotic bone impose a reduction in the freedom of stapedial movement. In a technical sense, the elasticity of the ossicular system is sharply increased at the oval window by this encroachment; moreover, this effect will continue to be the

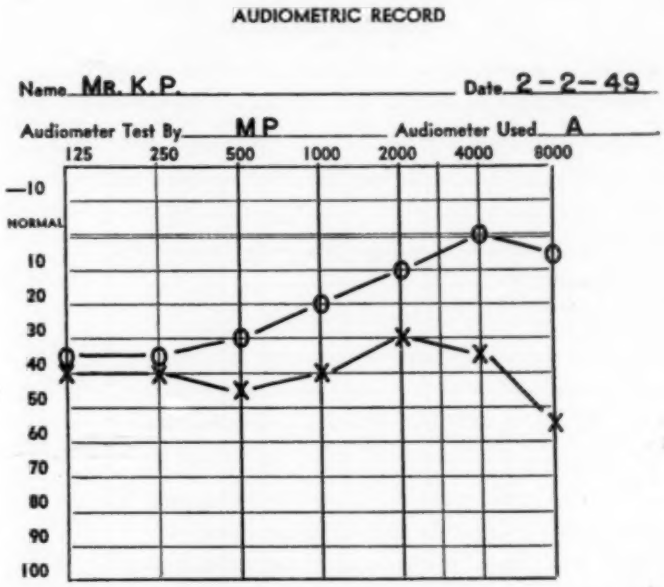


Fig. 4. Hearing levels for air conduction exhibited by a 51-year-old man having clinical otosclerosis without secondary nerve degeneration. The right ear, with incomplete stapes fixation, reveals loss at low frequencies suggestive of "stiffness" increase. The left ear, with complete stapes fixation, shows an additional loss at the high frequencies suggestive of "mass" increase.

main one as long as some stapedial motility remains. The mechanical state must change radically when fixation becomes complete. The stapedial footplate must now be so firmly locked within the oval window that the stapes is loaded, at least to some degree, by the mass of the skull. This increased mass should reduce the capacity of the system to transmit high frequencies. One notes, in Fig. 4, that the added im-

pairment exhibited by the left ear, where the stapes was completely fixed, is of this very nature.

The foregoing discussion is schematic, but it illustrates the manner in which awareness of physical concepts can enhance the clinician's evaluation of the hearing losses he observes. A further illustration, with completely different practical

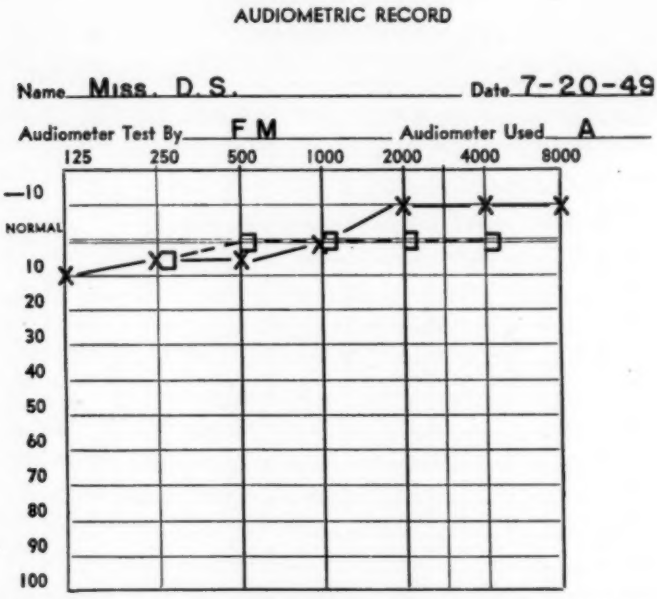


Fig. 5. Hearing levels for the "normal" left ear of a 37-year-old woman having clinical otosclerosis in the right ear. The tilt of the air conduction curve in relation to the horizontal curve for bone conduction illustrates earliest audiometric clue to the onset of stapes fixation.

implications, is to be found in the audiometric configuration of pre-clinical otosclerosis. This configuration was suggested by Campbell.[2] Graham[7] found the configuration to be common among teenagers and young adults whose hearing was "normal" and who had close relatives exhibiting otosclerosis. The configuration is illustrated in Fig. 5. It is characterized by a tilting of the air conduction curve without concomitant

shift in the bone conduction response. The tilting is sufficient so that an air conduction loss of 10 to 15 db. appears at 125 cycles. Hearing is near normal at 1000 and 2000 c.p.s. Hyperacuity appears at still higher frequencies. This tilting, when interpreted from the standpoint of impedance concepts, leads one to suspect increased stiffness within the conductive system. As already pointed out, the mechanical effect of incomplete stapes fixation is to increase the stiffness of the system.

The present instance represents a condition where the fixation process is so minor that hearing loss of clinically noticeable magnitude has not yet resulted. Nonetheless, the audiogram shows a disturbance in responsiveness to air-borne stimuli which is attributable to minor changes in the conductive mechanism, and which is easily identified by the fact that the bone conduction curve does not reveal any evidence of this disturbance.*

The exciting feature of this configuration, from the medical point of view, is that it reveals the presence of otosclerosis early enough so that preventive measures can be applied once they are discovered. Thus, the time may come when clinical otosclerosis, as we know it today, will seldom be allowed to develop, because the disease can be discovered very early and can be controlled through medical, dietetic, or other non-surgical means.

In summary, as exemplified by the cases just discussed, lesions of the conductive system produce changes in hearing which are consistent with the principles governing mechanical systems. The clinician can facilitate his tasks in diagnosis by remaining alert to the manifestations of these principles which appear in the air conduction audiogram.[11]

VIII.

The problem of differentiating among sensori-neural losses is complicated. One task is to segregate cochlear (end-organ) lesions from retro-cochlear (neural) lesions. A further task

*The mechanical effects are so minor at this stage that the notched depression of the bone curve which one expects with clinical otosclerosis has not yet appeared.

is to locate the site of damage accurately, to designate definitively the structures involved, and to describe specifically the pathology which is, or has been responsible for the hearing difficulty. Of course, much needs to be learned before full diagnostic precision will become possible here.

Fortunately, a wide variety of auditory tests directed toward this goal have been developed or adapted in recent years. Many of these tests have been of the supra-threshold variety; *i.e.*, loudness balance techniques, methods for measuring DLs for intensity change, tests of speech discrimination in quiet and in noise, and the delayed side-tone procedure. It is still too soon to specify the ultimate usefulness of all these techniques; however, their clinical applications are rapidly becoming clarified in consequence of extensive research from different parts of the world. The diagnostician's efficiency has already been enhanced significantly, and future research will bring him added insight.

The emphasis on new tests has diverted attention from the contribution which conventional threshold audiometry can make to differential diagnosis among sensori-neural lesions. There is no reason to believe that the limits in interpretation of the audiogram have been reached; in fact, critical study of audiometric configurations suggests subdivisions in diagnostic categories which the clinician does not currently consider. Clinicians, (and theorists) can profitably re-examine the significance of threshold audiometry.

IX.

It would be premature to attempt a systematic description of the array of clues to differential diagnosis which are available through plotting thresholds for pure tones. One can, however, illustrate the existence of such clues by discussing examples involving sensori-neural losses. These examples consist of audiometric pecularities which are easily recognized, but which still await full interpretation.

One example is the configuration of sensori-neural loss wherein acuity for low frequencies is near normal, and wherein there is sharp transition to a plateau of high frequency

loss at about 60 db. This type of impairment is relatively common, and its existence can be demonstrated either from contemporary clinical records or from the literature. Figs. 6 and 7 are samples of this configuration taken from Bunch's original records, which are preserved at Northwestern University. Fig. 8 plots a similar impairment, as found in a

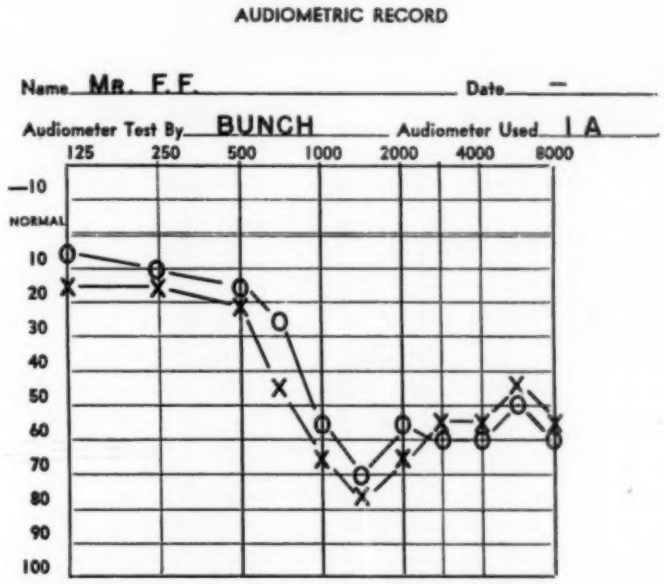


Fig. 6. Hearing levels for air conduction exhibited by 44-year-old man employed as a riveter.

case tested at Northwestern in 1957. These three exemplify an audiometric feature which is quite common, even though it does not repeat itself with perfect consistency from one patient to the next. The existence of a distinctive type of lesion can be postulated from the fact that so many high frequency losses plateau at approximately the 60 db level of loss. This lesion must be of such nature that it does not interfere with acuity for low tones, nor with response to intense sounds at high frequencies. One can not help but

speculate that this lesion may be geographically restricted, so that neural representation from the outer hair-cells within the basal region of the cochlear spiral is lost, while excitation from the inner hair-cells within the same region continues to have central representation, as does excitation from the more apically located hair-cells of both varieties. Minor differences in the extent, the severity, and the purity of the lesion could

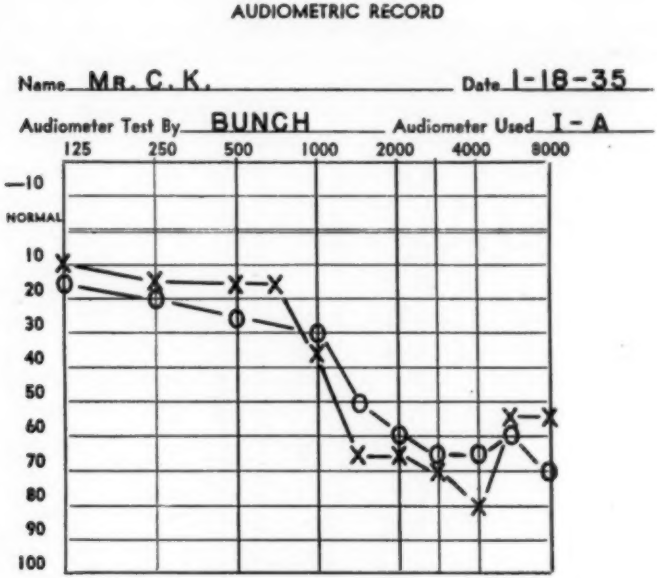


Fig. 7. Hearing levels for air conduction exhibited by a 25-year-old man with sensori-neural involvement of unspecified etiology.

account for the clinical variations observed in hearing losses of this general type.

The foregoing speculation does not require the presumption that the outer hair-cells have been destroyed, although such a situation is one possibility. An equally tenable alternative is the possibility that the innervation from these hair-cells to the central nervous system is defective. There may, moreover,

be other alternatives, and subsequent study may reveal that several types of lesion produce the audiometric configuration under discussion.

The idea being stressed at the moment is that this unique audiometric configuration must result from patterns of lesion which are distinctive. It is convenient, since the external

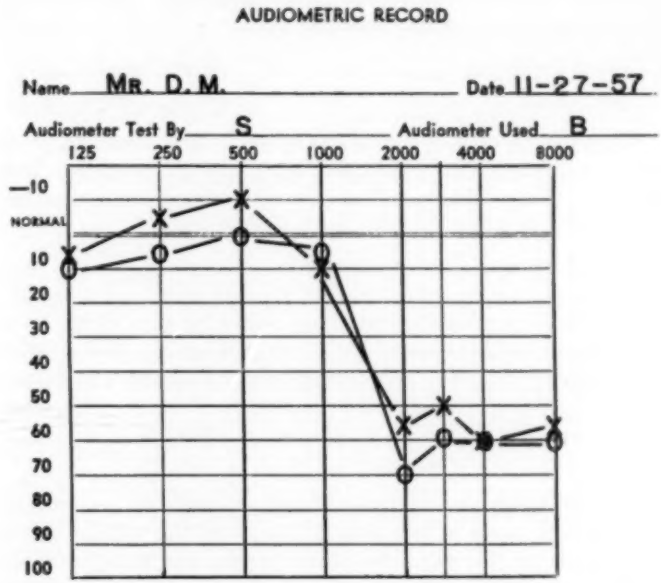


Fig. 8. Hearing levels for air conduction exhibited by a 59-year-old woman with bilateral sensori-neural involvement of unknown etiology.

hair-cells are thought to mediate response to weak stimuli and are more susceptible to damage from intense stimulation,[6] to evoke the speculations advanced in the preceding paragraph; however, these speculations are not essential in the present discussion. They merely illustrate the kind of thinking which study of threshold audiograms excites. The fundamental premise, which neither clinician nor theorist dare

ignore, is that every hearing loss is the product of a specific set of lesions. These lesions determine the nature of the audiometric configuration which the patient exhibits. The fact that diverse patterns of pure tone loss are encountered indicates that auditory lesions differ not only in extent but also in kind. One way in which clinical audiology can participate in classifying the conditions involved is by renewing its interests in the details of audiometric configuration. One practical benefit will be that the diagnostic usefulness of conventional audiometry will increase.

X.

Consider two additional illustrations of the diversity of information which the pure tone audiogram can contain. Both illustrations involve peculiarities of auditory function frequently found in conjunction with presbycusis.

One of these peculiarities is exemplified in Fig. 9. Note that here the loss exhibited at 125 c.p.s. is greater than at 250 c.p.s. This configuration has been observed as a group trend in various studies where general populations of elderly people have been sampled.[4,12] It also appears as a clearly identifiable feature in the audiograms of many clinical cases, who sometimes yield thresholds 15 db. poorer at 125 c.p.s. than at 250 c.p.s.

Such findings are baffling if one attempts to account for them on the basis of peripheral lesions in the sensori-neural mechanism of the ear. In other words, the peripheral degeneration of hair cells and spiral ganglion cells, which has been observed to characterize presbycusis,[18] cannot account for the increased loss at 125 c.p.s. This degeneration affects the sensori-neural structures serving the basal region of the cochlea. It consequently disturbs acuity for higher frequencies. Any effect it may have at 125 c.p.s. should not be more severe than the effect at 250 c.p.s.; moreover, these two latter frequencies are within the range which cochlear agitation is diffuse and where, stated in general terms, the same structures will be stimulated by both stimuli. Thus, since volleys of neural impulses are the intermediaries to perception

of low frequency tones, the distinction within the auditory system between a tone of 125 c.p.s. and one of 250 c.p.s. is a difference in the rate at which the volleys are triggered.

It seems reasonable to conclude that many elderly persons who retain equivalent peripheral sensitivity at these two frequencies require a greater comparative intensity in order for

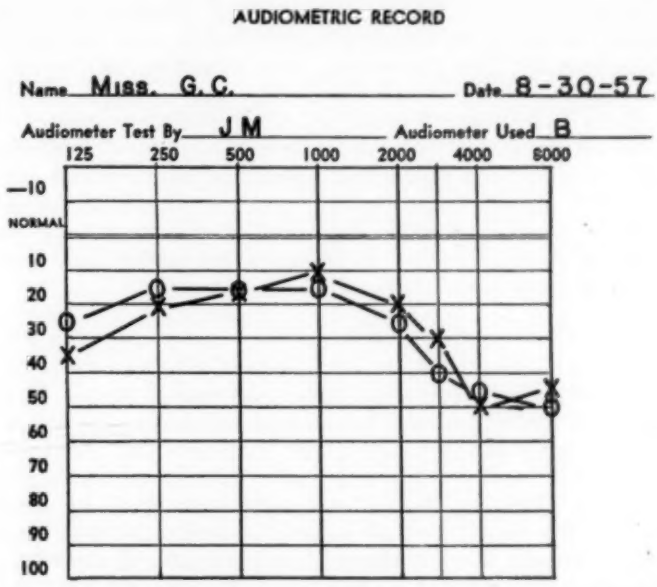


Fig. 9. Hearing levels for air conduction exhibited by an 80-year-old woman having presbycusis. This chart exemplifies cases with greater loss at 125 c.p.s. than at 250 c.p.s.

stimulus to be perceived when the volley rate is 125 per second, than when it is 250 per second.* In such event, the extra impairment in acuity found at 125 c.p.s. must depend upon a disturbance of function within the central nervous

*The alternative would be that the ageing of structures within the inner ear changes the mechanical constants of the auditory system in such manner that its responsiveness to very low frequencies is differentially impaired. This possibility seems a less likely source for the phenomenon under discussion.

system. It would be foolhardy to speculate regarding the site or the nature of such malfunction; however, because such a disturbance could disrupt various facets of auditory behavior[1,17] and hence complicate a patient's problem, the clinician must be alert to the potentiality that it may be present in a particular case. Proper attention to the audiometric configuration can warn the clinician of this potentiality.

A second peculiarity in the auditory response of some elderly patients is an abnormally great loss by bone conduction at 500 c.p.s. This phenomenon was identified by Bernero, who noted it in the records of the Hearing Clinics at Northwestern University. The audiometric technique employed in these Clinics is one wherein masking is not used routinely while testing bone conduction. Masking is introduced only when the patient states that his auditory experience is not localized to the ear under test. This fact is mentioned because, as described below, it appears to be an important condition in evoking the Bernero phenomenon.

A typical example of the phenomenon is reproduced in Fig. 10, which gives results for only one ear. The agreement between air and bone thresholds is good except at 500 c.p.s. Tradition would lead one to discount the difference at 500 c.p.s. as being due to testing error; however, this particular discrepancy is observed so repeatedly that it can not be dismissed in a cavalier fashion, and one can not ignore evidence such as the observation by Ciocco,[5] reported in 1934, that elderly persons have disproportionately reduced acuity by bone conduction when tested with the 512 c.p.s. tuning fork; therefore, the clinician is justified in suspecting that he is encountering the Bernero phenomenon when the patient is elderly, when the bone conduction threshold of 500 c.p.s. is 10 to 15 db poorer than the air conduction threshold, and when the remainder of the audiogram shows the close correspondence between measures to be expected in sensori-neural loss.

The Bernero phenomenon cannot be explained by conventional principles of audiometric interpretation. These principles specify that loss is less for bone than for air when

the impairment is of the conductive variety, whereas the two losses are equal when the lesion is a sensori-neural one. Only in rare instances of skull anomaly, such as Guild has described,[8] could gross mechanical factors account for excessive loss by bone, and here the effect should not be limited to one frequency; thus, only two alternatives remain.

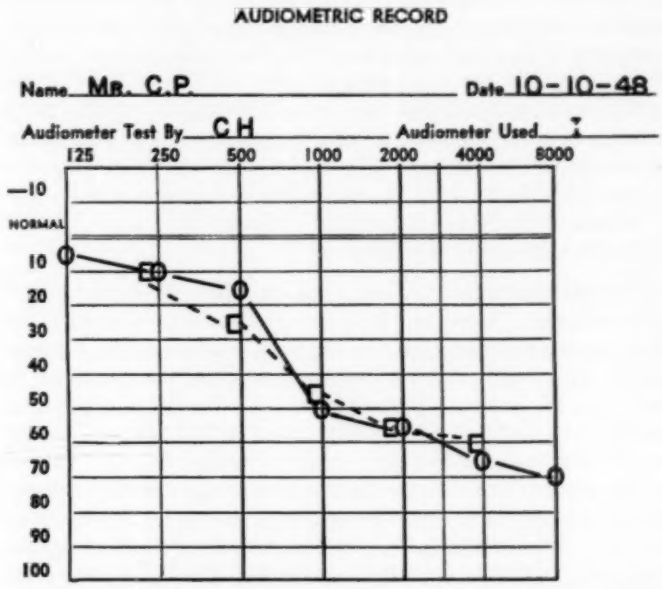


Fig. 10. Hearing levels for the right ear of a 71-year-old man having presbycusis. This chart illustrates cases with noticeably greater loss at 500 c.p.s. by bone conduction than by air conduction.

One possibility is that ageing has modified the mechanical constants within the inner ear in such a manner that a differential reduction in the responsiveness of the auditory system of skull-borne vibration at 500 c.p.s. has been produced. This possibility seems relatively unlikely, even though some writers have postulated that ageing sometimes produces a conductive loss within the inner ear in consequence of the physical changes that occur there.

The alternate possibility is that the Bernero phenomenon appears whenever the bone conduction stimulus is more difficult to perceive than is the air conduction stimulus. This alternative presumes that peripheral sensitivity is the same for the two modalities, but that a functional peculiarity is operative which makes conscious recognition of the bone conduction stimulus at 500 c.p.s. difficult. A potential mechanism comes to mind quickly. One need merely remember three facts: *1.* in presbycusis the two ears usually exhibit bilateral symmetry of loss; *2.* when such bilateral symmetry exists the air conduction stimulus activates only the ear under test, so that monaural excitation occurs at threshold; *3.* the bone conduction stimulus reaches both cochleas. Such a stimulus should excite both ears if they are similar in sensitivity and neither is masked. True, the levels of stimulation will seldom be identical, and the experience of hearing will ordinarily localize to one ear or the other.

Bilateral excitation by bone goes unnoticed in most clinical circumstances; however, it may be a critical consideration when perceptual abilities are reduced, albeit subtly, by age. Stated differently, the foregoing considerations lead one to interpret the Bernero phenomenon as indicating a condition wherein the cortical differentiation of two trains of neural impulses is more difficult when the impulse rate is 500 per second, than is the differentiation of a single train; and one must presume, since the phenomenon is restricted to 500 c.p.s., that this disadvantage in bilateral hearing is not apparent when the stimulus frequency is either markedly lower or markedly higher in frequency. The reasons for such a circumscribed disturbance in perception are obscure, but its existence cannot be ignored. Thus, it would seem that the Bernero phenomenon is a clue to a central perceptual disturbance. As such, it is a second audiometric aid to the identification of those elderly persons whose hearing difficulties are not exclusively of peripheral origin.

XI.

A point worthy of re-emphasis is the fact that understanding of auditory abnormalities is currently imperfect. Both

the clinician and the theorist must remember that there are aberrations in hearing function which the usual testing procedures do not reveal. The diagnostic significance of such aberrations will emerge as the aberrations become known and understood. One result will be further insight into the meanings which can be assigned to results of conventional audiometry.

Consider the phenomenon of abnormal auditory adaptation as it is revealed through the tone decay test.[3] The procedure is to present a sustained pure tone at threshold for a full minute unless the patient signals that the tone has become inaudible. In the latter event, the intensity is increased 5 db., and the timing is started anew. This routine is repeated until an intensity is found at which the tone remains audible for 60 seconds. The exploration is then complete at this frequency, and a new exploration may be started at another frequency.

Results for the tone decay tests are classifiable into three general categories. Cases with conductive loss, and some with sensori-neural impairments, do not reveal auditory adaptation at threshold. Other cases of sensori-neural loss exhibit moderate adaptation for high frequency tones, *i.e.*, up to 25 db. drift in the level at which the tone remains audible. Adaptation for low frequencies is rare. Finally, occasional instances occur in which adaptation is so extreme that the tone is heard for a full minute only at great intensities, or else not at all. This extreme type of adaptation has been encountered both in cases where there is hearing loss in the ordinary sense, and in instances where standard audiometry yields normal thresholds. Such findings indicate unusual auditory disturbances. Tentatively, it appeared that adaptation as extreme as this is the result of a neural lesion,[12,10] while moderate adaptation probably has its origin in the cochlea.[9] It would seem, moreover, that not all types of disturbance in either locus produce abnormal adaptation at threshold, since many sensori-neural losses do not exhibit any tone decay at threshold. This matter will have to be clarified by further research.

The foregoing discussion not only illustrates the fact that abnormalities in auditory function are complicated and, hence,

emphasizes the need to classify clinical sub-types more adequately. It also calls attention to phenomena which must be kept in mind by the clinician as he interprets conventional threshold tests. He should, for example, become immediately suspicious that a problem of abnormal adaptation may exist whenever he encounters a subject whose threshold level seems to vary excessively from moment to moment. Stated differently, the fact that a patient's audiometric responses are highly unreliable may be a clue to unique features in his hearing loss which can be quantified if explored with an appropriate additional test.

XII.

In summary, the pure tone audiogram can be of great help in the differential diagnosis of hearing losses. The configuration of the air conduction curve often suggests etiology, and in cases of conductive loss, the nature of the mechanical disturbance. In addition, air-borne relationships distinguish conductive from sensori-neural involvements. Minor variations in the bone conduction curve are characteristic of different conductive lesions. Finally, threshold audiograms contain much information which awaits full codification and which, when so codified, will contribute to a new level of precision in differential diagnosis among hearing losses.

REFERENCES.

1. Bordley, J. E., and Haskins, H. L.: The Role of the Cerebrum in Hearing. *Ann. Otol., Rhinol., Laryngol.*, 64:370-382, 1955.

2. Campbell, P. A.: The Importance of the Impedance Formula in the Interpretation of Audiograms. *Trans. Amer. Acad. Ophthalmol. Otolaryngol.*, 54:245-252, 1950.

3. Carhart, R.: Clinical Determination of Abnormal Auditory Adaptation. *Arch. Otolaryngol.*, 65:32-39, 1957.

4. Carhart, R.: "Progress Report on Auditory Impairments of Old Age." Hearing Clinic, Northwestern University, 1957.

5. Ciocco, A.: Disproportionate Shortening of Bone Conduction. *Acta Oto-Laryngol.*, 22:529-539, 1935.

6. Davis, H.: Biophysics and Physiology of the Inner Ear. *Physiol. Reviews*, 37:1-49, 1957.

7. Graham, A. B.: "An Audiological and Otological Investigation of Normal Hearing Individuals with a Family History of Otosclerosis." Doctoral Dissertation, Northwestern University, 1953.

8. GUILD, S. R.: Hearing by Bone Conduction: The Pathways of Transmission of Sound. *Ann. Otol., Rhinol., Laryngol.*, 45:736-754, 1936.

9. HOOD, J. D.: Auditory Fatigue and Adaptation in the Differential Diagnosis of End-Organ Disease. *Ann. Otol., Rhinol., Laryngol.*, 64:507-518, 1955.

10. JOHANSEN, H.: Relation of Audiograms to the Impedance Formula. *Acta Oto-Laryngol.*, Supp. 74:65-74, 1948.

11. KOBRAK, H.: Impedance of Sound Conduction System. "Otolaryngology." (Ed. Coates, G. K.; Schenck, H. P., and Miller, M. V.). Prior & Co., Vol. 1, Chapt. 3:53-59, Hagerstown, Md., 1955.

12. KLEEMEIER, R. A., and JUSTISS, W. A.: Adjustment to Hearing Loss and to Hearing Aids in Old Age. "Aging and Retirement." (Ed. Webber, I. L.). University of Florida Press, 34-48, Gainesville, Fla., 1955.

13. KOS, C. M.: Auditory Function as Related to the Complaint of Dizziness. THE LARYNGOSCOPE, 65:711-721, 1955.

14. LIERLE, D. M., and REGER, S.: Experimentally Induced Temporary Threshold Shifts in Ears with Impaired Hearing. *Ann. Otol., Rhinol., Laryngol.*, 64:263-277, 1955.

15. LIERLE, D. M., and REGER, S.: Correlations Between Air and Bone Conduction Measurements with Wide Frequency Ranges in Different Types of Hearing Impairments. THE LARYNGOSCOPE, 56:187-224, 1946.

16. METZ, D.: The Acoustic Impedance Measured on Normal and Pathological Ears. *Acta Oto-Laryngol.*, Supp. 63:1-254, 1946.

17. PESTALOZZA, G., and SHORE, I.: Clinical Evaluation of Presbycusis on the Basis of Different Tests of Auditory Function. THE LARYNGOSCOPE, 65:1136-1163, 1955.

18. SCHUKNECHT, H.: Presbycusis. THE LARYNGOSCOPE, 65:402-419, 1955.

DR. HIRSH: We have time for a question.

DR. MOE BERGMAN, (New York, N. Y.): I would like to express my own gratification, as I am sure the whole group does. Professor Carhart is continually urging us to go back and look at our audiograms very carefully.

I would like to ask a question on that last slide, and that is on that very provocative case of tone decay. I wonder, again, whether there is a relationship between the recruitment and the loss of loudness in the better ear as against the growth of loudness in the worse ear. Is the recruitment due to a growth of loudness in the poor ear, therefore, giving us equal loudness, or because of the decay in the better ear?

DR. CARHART: There might be just the reverse. The better ear has such a low level of loudness that the match is made close to threshold even though there is no recruitment, in the conventional sense, in the better ear.

These questions which you raise are very exciting and represent part of the speculation which ought to be done. I can simply report to you facts. In elderly people, at least, I am convinced that this phenomenon of poor hearing, 125 and 250, is sufficiently frequent so that it cannot be accounted for on mechanical ground or some immediately explainable ground which we might think of, since we do not find it simultaneously in the same clinical run, so to speak, on cases that do not fall in this category.

DR. HIRSH: The first question had to do with physical factors that make the measurement of 125 unreliable.

DR. CARHART: I thought I answered that simply by stating that this is an observation made under clinical circumstances where other types of cases did not seem to show unreliable responses to 125.

DR. TAUNO PALVA, (Turku, Finland): I should like to comment on certain aspects of Dr. Carhart's paper, although I am supposed to discuss some of these phenomena in my own presentation to be given later.

The last slide shown by Dr. Carhart described a very large stimulatory adaptation loss during only some thirty or forty decibels of stimulation. What is even more remarkable is that there was similar fatigue in the healthy ear as in the deaf ear. If the result is real, I think it is an extreme variant. I have made a continuous threshold recording during four minutes stimulation in over two hundred cases, representing mostly recording perceptive type of lesions, but I have never seen as severe per-stimulatory fatigue as described by Dr. Carhart.

In my experience the threshold sensitivity loss does not generally exceed 30 to 40 db, and it does not occur in more than 15 to 20 per cent of the patients with perceptive lesions.

My second point concerns the slight loss of bone conduction observed by Dr. Carhart at higher frequencies in certain cases with chronic ears. Supposing that some of these ears had perforations in the tympanic membrane with dry cavity, I should like to know whether bone conduction measurements

were also made when the tympanic perforation was closed, for instance, with a cigarette paper patch. The possible difference in the open and closed perforation could tell us whether the slight bone conduction loss was due only to the altered conductive ability of the tympanic membrane or whether there were also changes in the window nucleus that could be responsible for a covered bone conduction.

DR. CARHART: I would not wish to attempt to state what particular kind of mechanical factor might be responsible for the type of thing I was talking about in the bone conduction test.

I will comment briefly on the first point you made, Dr. Palva. This particular kind of a test is a sort of first inventory, of course, and this particular case, while it is extremely unusual, and I suppose, for that reason makes a beautiful demonstration, is nonetheless one whose validity, whose clinical reality I cannot doubt; because the person who did this happened to be a Danish physician who was working with us at the time, and who did not believe the results of the test the first time and, therefore, returned and repeated the test and got the same results the second time.

DR. HIRSH: Our discussion of audiometry has begun with the role of the audiogram, which is, as Dr. Carhart told you, useful, and in addition, the pure tone adaptation test which Dr. Carhart has implied will be useful in the future.

Let us return now to a consideration of a very important set of problems surrounding the syndrome known as Ménière's disease.

MENIERE'S DISEASE.

THEODORE E. WALSH,

St. Louis, Mo.

I would like to bring up one point first about audiology in the diagnosis of Ménière's disease. I find the audiologist is quite a help, but I would emphasize that the diagnosis is made

by clinical observation. I am not being facetious; I just want to emphasize the point that it is a careful history of the case and a careful observation of the patient by which the diagnosis of Ménière's disease is made. The audiologic findings simply confirm the diagnosis.

Let us first take up the symptoms of Ménière's disease. Patients with this disease usually seek help because they are dizzy. It is usually not difficult to determine whether this dizziness is truly vestibular in origin or whether the so-called dizziness is not related in any way to the labyrinth. Although the main complaint is directed at the vestibular symptoms, it is really cochlear dysfunction which is responsible for the condition. I emphasize the point that I believe Ménière's disease is primarily a cochlear disease.

In taking a careful history of patients one can usually determine that they noticed some loss of hearing before they ever had their dizzy spells. Patients seeking help for Ménière's disease not only complain of dizziness but they also notice deafness, and they complain bitterly of tinnitus.

Now to discuss these symptoms individually, first, tinnitus. The most common description given of this tinnitus is that it sounds exactly like the noise heard when listening into a seashell, or the sound of the roar of the surf breaking on the seashore. Those individuals who have never been fortunate enough to listen into a seashell or hear the surf, will tell you that the sound they hear in their ear resembles the roar of a high wind in a forest or sometimes the sort of sound one hears when listening close to a telephone pole in a high wind. The patients also complain of a full feeling in the head which cannot be described in any other way except that it is a full feeling. The hearing fluctuates. At times these patients say they hear "perfectly" and at other times "nothing at all," but, on careful questioning about the hearing, most of them say they can hear but they do not understand. This ability to hear and not understand is easily demonstrated when talking to these people. One seldom has to raise one's voice in talking to a patient with Ménière's disease even if he is in a period of "hearing nothing at all," but one must talk very slowly and very distinctly.

Another complaint, which is more noticeable in musical people, is that of a different sensation of sound in the two ears, diplacusis. This is very common. If the patient is not musical he will tell you that sounds are different in the two ears, and he describes the sound in the worse ear as fuzzy. In the office we test for this diplacusis by the use of tuning forks, and I would emphasize that it is important to test with all forks throughout the whole range, because diplacusis may not be present at every tone. Frequently they notice no difference, for example, with a 500 cycle fork whereas when one uses the 4000 cycle fork it sounds different in the two ears and vice versa.

The vestibular tests are on the whole not much help in making a diagnosis of Ménière's disease. We may find a normal response in the two labyrinths, we may find a canal paresis in the affected ear, or we may find a directional preponderance, first to the affected side and later to the normal side. It depends a good deal on how long the condition has been present as to what vestibular findings will be. Now I would like to show some slides that illustrate these various points, the important points in the audiology of Ménière's disease.

Fig. 1—In this you see the pure tone audiograms of the two ears, one ear being perfectly normal and the other one not. This is not a very common pure tone curve in early Ménière's disease. As a rule the pure tone audiogram shows a greater loss in the low tones and almost no loss in the high tones in the early cases, and later the curve flattens and there is an equal loss throughout the scale; still later more and more of the high tones are lost as well as the low, so that one has a down-sloping curve.

Diplacusis is well shown in this individual, and you notice that the 500 cycle tone is matched fairly closely, but at 4000 cycles he matches it with 2890. I always feel sorry for people with that sort of diplacusis. It must be incredibly difficult to live with, if one is musical.

One of the points I mentioned, in our office examination, that these individuals had not much loss for speech at thresh-

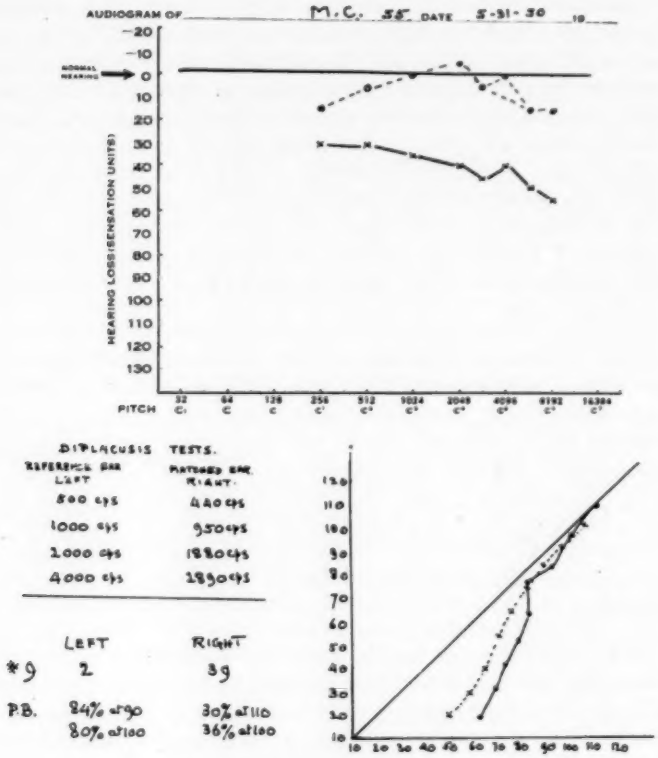


Fig. 1.

old but considerable difficulty with discrimination, and this is shown here. I would like to emphasize this point, because in going through a considerable number of these cases I found in no instance was the discrimination score better than 50 per cent, whereas the threshold was seldom more than a 40 decibel loss.

Figure 2 illustrates much the same thing. In this individual the fluctuation in hearing is well shown. I would like to emphasize this fluctuation in hearing because it makes it very difficult for us, as clinicians, to determine whether any

therapy we use is really doing any good unless we can follow the patient over a considerable period of time. This particular individual was under treatment during the time between December, 1950, and February, 1951, and it will be seen that his hearing was different on three different occasions. Here, in January, 1951, he has a normal hearing for speech, whereas in February, in spite of therapy, he drops to a 40 per cent discrimination score and a 29 decibel threshold loss.

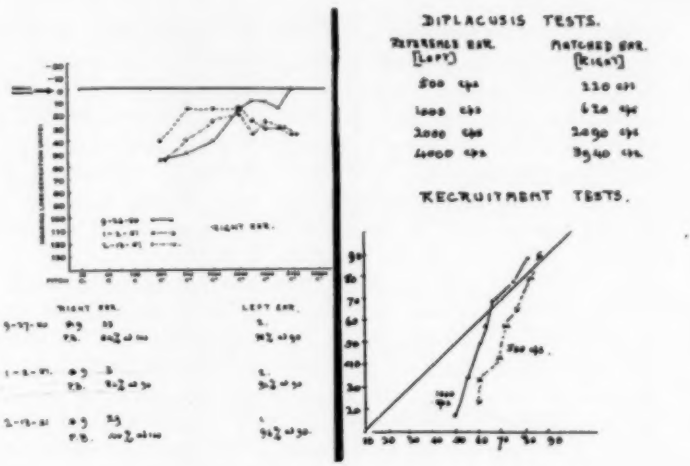


Fig. 2.

Figure 2 also shows the same patient's recruitment and the diplacusis. In this instance, he had a considerable difference in the hearing for low tones rather than in the high.

Now the fluctuation in hearing, I think, is important. It is very important to start therapy in these individuals when the hearing is fluctuating, because it seems to me that that is when there is a chance of saving some hearing and really rehabilitating these individuals. It is true that the treatment of Ménière's disease is directed first toward the relief of the unpleasant and dangerous vertigo. Unfortunately, that is

	Right								Left								Speech Right		Speech Left	
Date	256	512	1024	2048	2896	4096	5792	8192	256	512	1024	2048	2896	4096	5792	8192	T.L.	Dis.	T.L.	Dis.
12/1/55	45	55	65	50	50	60	/	/	25	30	15	10	45	50	65	60	85	6%	16	80%
12/29/55	40	50	60	35	50	65	/	/	30	40	40	25	50	55	60	65	....	....	....	....
1/9/56	30	35	35	30	55	65	/	/	45	40	45	20	50	55	65	60	27	32%	29	54%
2/6/56	25	30	25	15	40	65	/	/	30	45	50	30	50	55	65	60	15	64%	43	24%
2/20/56	....	....	....	....	....	....			....	....	....	....	....	....	....	....	16	72%	30	50%
3/5/56	25	30	20	15	45	65	/	/	20	5	15	20	50	55	65	60	12	66%	0	80%

The meaning of the abbreviations is as follows: T.L., threshold loss; Dis., discrimination.

Fig. 3.

where it usually stops. As a matter of fact, I don't believe that the relief of vertigo is nearly so difficult as the preservation of hearing. That this preservation is possible by treatment I would like to show in Figure 3.

This is a man who has a bilateral Ménière's disease. You can see that his hearing in both ears is fluctuating considerably but on treatment he has remained at this good level for quite some time.

Figure 4 illustrates the same sort of fluctuation. This is an old man with presbycusis in one ear and Ménière's disease in the other. When we started therapy the hearing in the ear with Ménière's disease had only 28 per cent discrimination; but on treatment it went up to 76 per cent, and his threshold also improved. When the medication was changed his hearing dropped, but we have been able now to preserve him at a level of some 70 per cent discrimination for several years.

To summarize the audiologic findings, the important ones are. *1.* Fluctuation in the hearing which gradually becomes worse until almost complete deafness results. *2.* The loss at threshold for speech is not great in comparison with the discrimination loss. *3.* Early pure tone audiograms show an up-sloping curve and later the curve flattens and finally the high tones are lost. *4.* Diplacusis is almost always present. *5.* The tinnitus is described as a roaring sound and there is a full feeling in the ear. *6.* Finally, the most important point in the differential diagnosis of Ménière's disease is the invariable presence of recruitment that even becomes in some instances hyper-recruitment.

DR. HIRSH: Are there any comments?

UNIDENTIFIED DOCTOR: I would like to comment on diplacusis which is observed in many cases. In effect, the diplacusis may sometimes make very difficult the evaluation of the other ear, which is very useful for diagnostic purposes; and I should like also to stress that in my opinion it is not always easy to make a pure audiological diagnosis

	Right								Left								Speech			
																	Right		Left	
Date	256	512	1024	2048	2896	4096	5792	8192	256	512	1024	2048	2896	4096	5792	8192	T.L.	Dis.	T.L.	Dis.
2/19/53	0	0	0	15	30	35	25	25	25	20	10	45	50	40	65	45	....	....	....	....
3/25/53	0	0	0	30	45	50	50	55	50	35	45	35	45	50	50	45	....	....	....	....
5/5/53	0	0	0	45	55	55	55	60	45	35	30	25	45	45	55	35	11	66%	42	28%
2/9/54	0	0	–5	40	55	55	60	60	20	25	10	15	40	45	55	40	5	60%	16	76%
2/1/55	–5	–5	0	35	50	55	50	55	30	35	35	10	45	45	55	40	8	68%	28	72%
10/18/55	....	....	....	....	....	....	....	....	....	....	....	....	....	....	....	....	6	70%	30	54%
12/13/55	....	....	....	....	....	....	....	....	....	....	....	....	....	....	....	....	6	64%	31	56%
2/7/56	....	....	....	....	....	....	....	....	....	....	....	....	....	....	....	....	3	70%	27	60%

The meaning of the abbreviations is as follows: T.L., threshold loss; Dis., discrimination.

Fig. 4.

of Ménière's disease. We have several diseases characterized by vertigo which are not Ménière's disease, but are due to some disease of the nerves, for instance, the fact of a patient having vertigo and practically normal hearing. This could be an unusual case of Ménière's disease without impairment of the auditory function, but we could not do without the vestibular examination in all cases of Ménière's disease because it not only informs us about the condition of the vestibular apparatus, but also adds very much to our diagnosis. In effect, if you find a man who has vertigo and normal hearing and you send him to the audiologist who says that he finds a total loss—we are clearly dealing, not with a Ménière's disease but with a neuritis of the acoustic nerve, because in Ménière's disease it is impossible to have a total loss of function of the vestibular apparatus. On the other hand, we have total loss of hearing or a severe loss of hearing with a fairly normal vestibular nerve. Now, this is generally due to the fact that we are dealing with a cochlear neuritis. In fact, we might rely on some combinations of cochlear and vestibular diagnostic testing. If we have a total loss of hearing and a total loss of vestibular function on one side, we may, likewise, make a diagnosis of neuritis of the VIIIth nerve, and in the history of the patient we never discover any sign of even the slightest vertigo. We are confronted in this case with a tumor of the acoustic nerve. It is my opinion that no diagnosis of Ménière's disease should be made unless the vestibular examination and the cochlear examination agree as to their results. The results must go hand in hand, otherwise we are not certain that we are dealing with a Ménière's disease and not with an infection of the head nerves.

DR. VICTOR GOODHILL: I fully agree with Dr. Walsh that tinnitus may be the consideration that he described in Ménière's disease; however, I have seen this type of tinnitus in otosclerosis not too rarely, and in other lesions, which prompts me to urge my audiological colleagues here today to give more attention to tinnitus analysis in the overall approach to diseases of the ear. I think we have neglected the symptom of tinnitus, and that can help us a great deal if we set out to get some technique for clinical attention, qualitatively and quantitatively.

COCHLEAR VS. RETROCOCHLEAR LESIONS.

TAUNO PALVA,
University of Turku,
Turku, Finland.

It is a general rule that the major division between hearing disorders of conductive and perceptive type is made on the basis of a pure tone audiogram. If bone conduction is normal or clearly better than air conduction, the case is labeled as conductive; if it equals or is poorer than air conduction, the deafness is classified as perceptive. The first conclusion always holds true. In the latter case, classification is not so simple; therefore I feel that, before discussing the differences in the subgroups of perceptive deafness, it is necessary to say a few words about cases where air conduction approaches or equals bone conduction, though there is a purely conductive deafness.

Together with Ojala,[1] I have pointed out that in nearly one-third of the cases with simple acute otitis media, bone conduction is abnormally poor as long as the middle ear has not healed. In these cases, however, the diagnosis can be ascertained simply by looking at the tympanic membrane; but diagnosis is no longer so easy in cases of serous or adhesive otitis generally associated with only minor alterations in the tympanic membranes. These cases present a typical curve of perceptive component with lowered bone conduction, most severely at the higher end of the scale. The reason for this is either serous secretion which fills the window niches and the middle ear, or adhesions and thickened mucous membranes which fix both windows. In the absence of inflammation both air and bone conduction instantly return to nearly normal values when the serous fluid is aspirated.

Otoscopic examination with Siegle's speculum and aspiration of the middle ear are the most important assets in separating these cases from true perceptive deafness. In rare cases even exploration of the middle ear may be necessary to obtain a correct diagnosis and to return good hearing.

If it is established that the middle ear is normal, equal air and bone conduction indicate perceptive deafness. In the large majority of cases the lesion is localized to the end-organ or to the VIIIth nerve; only a few cases present a more central pathology.

Recruitment Testing.

In dividing perceptive deafness into these two main subgroups, it has been customary to use the recruitment of loud-

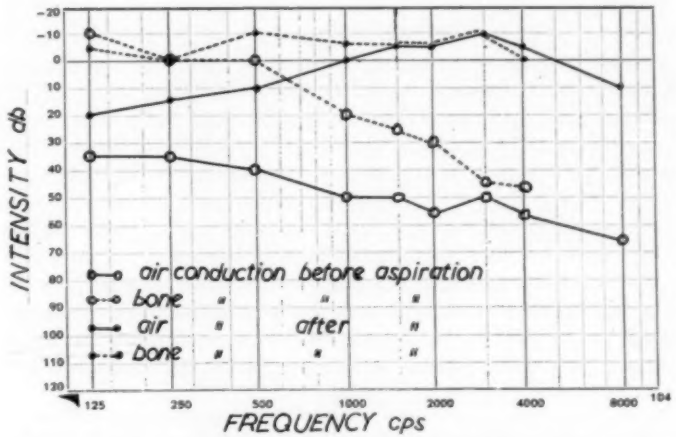


Fig. 1. A case with serous otitis media. Bone conduction is considerably lowered at higher frequencies but returns promptly to normal after aspiration of the middle ear.

ness as a criterion. Fowler's[2] original concept was that this phenomenon occurs in perceptive deafness in general, and is not typical of any more definitely localized lesion. Tests of loudness recruitment have enjoyed increasing popularity only since 1948 when Dix, Hallpike and Hood[3] stated that recruitment is pathognomonic of end-organ disease, more specifically of lesions in the hair cells.

The first histological correlation of loudness recruitment to the changes in the end-organ was obtained from cases of

Ménière's disease where the special feature, agreed upon by all investigators, was distension of the cochlear duct. Special damage to the hair cells was observed by Hallpike and Cairns[4] and by Dix and Hallpike[5] in a total of three ears. Other workers,[6-8] however, were not able to find such hair cell changes as could be definitely separated from postmortem degeneration. On the other hand, it was common observation that ganglion cells and nerve fibers remained intact at least in the earlier part of this disease, and thus the presence of recruitment must necessarily be related to the structures peripheral to the spiral ganglion.

Another histological correlation comes from the animal studies of artificial acoustic trauma.[9-11] It is agreed that the lesions are primarily seen in the mesothelial cells of the tympanic lamella and in the external hair cells. Following very intense stimulation the whole organ of Corti may be destroyed. The lesion is also evidenced by a marked reduction of the cochlear potentials, although such a reduction may occur in ears without demonstrable histological trauma.

In human material, exposed to acoustic trauma, the resulting lesions regularly exhibit recruitment phenomenon. This is the case in experimentally induced hearing losses by stimulation with intense sounds[13] and in the ears subjected to occupational acoustic trauma.[14]

The third histological correlation is derived from Saxén's studies of presbycusis.[15] Among his 44 cases there were 13 in which the lesion was restricted to the spiral ganglion and nerve fibers and the end-organ itself was intact. Another main group comprised 19 persons in which there were severe angiosclerotic changes in the stria vascularis and high grade degeneration of the organ of Corti, especially in the external hair cells; but sometimes the sensory structure as a whole was degenerated in certain areas. As a rule some reduction in the spiral ganglion cells and in the number of nerve fibers also occurred in these cases. In this angiosclerotic group Saxén obtained anomalous results with high intensity Weber tuning fork tests: lateralization to the ear showing greater changes in the organ of Corti. At the time of Saxén's clinical

investigation little was known about the recruitment phenomenon, but we now know that this type of result of a Weber test is one way to demonstrate recruitment phenomenon.[16]

The presence of recruitment in part of the presbycusis cases is easily demonstrated with the monaural loudness balance test. Pestalozza and Shore[17] reported complete recruitment in four cases out of 20 studied. It is also the experience at our laboratory that many presbycusis ears show recruitment.

We must now consider closely the group of neural deafness showing prominent lesions in the nerve fibers and also in the spiral ganglion, *viz.* acoustic neurinomas. Dix, Hallpike and Hood,[3] who first described this group as typical non-recruiting deafness, later[18] found recruitment of loudness in 12 per cent of the tumors. Even earlier, defending his theory that the recruitment phenomenon is typical of perceptive lesions in general, Fowler[19] described one case with complete recruitment. I have recently seen a case of cerebellopontine angle lesion with recruitment. The explanation for recruitment in these typical neural lesions has been assumed to lie in the disturbed cochlear blood supply, which adds end-organ damage to the picture. Dix and Hood[18] even described two cases in which there was recruitment prior to operation when the cochlear blood supply had presumably returned to normal.

In the presbycusis group of Saxén,[15] showing only spiral ganglion cell and nerve fiber atrophy, the clinical evaluation showed no evidence of the presence of recruitment.

Evidently one can safely say that, if there is recruitment, there is also an end-organ lesion, and that in a pure ganglion cell nerve fiber atrophy there is no recruitment; but there remain a number of cases in which both lesions are combined and recruitment may or may not be present. It is probable that if the end-organ lesion dominates, but is not too severe, there is recruitment, and if the neural lesion dominates, there is none. When these conclusions are applied to the audiological findings, we must say that positive recruitment is evidence of an end-organ lesion, but does not necessarily exclude an associated neural involvement. Contrariwise, the absence

of recruitment means that there is a dominating neural lesion but it does not exclude a simultaneous end-organ damage.

At present we do not exactly know why recruitment occurs in end-organ lesions, although many hypotheses have been advanced to explain the phenomenon. It is commonly believed that as the excitation becomes stronger, more and more hair cells are activated, especially internal hair cells, which are more resistant to damage than the external ones. The resulting initial discharge would then be capable of stimulating a normal amount of nerve fibers, so that a normal loudness sensation results; however, the matter is not so clear as that. If one believes that the cochlear potentials are a fair indicator of the condition of the hair cells and responsible for initiating the nerve impulses in the cochlear nerve endings, one is at a loss to understand the origin of the recruitment phenomenon.

In Fig. 2, which is reproduced from Wever and Laurence's recent book[20] "Physiological Acoustics," we notice first a normal cochlear potential curve obtained by increasing the stimulus and recording the increase in the potentials. The lower curve describes a similar response from an ear subjected to acoustic trauma, and it shows a constant deviation from the upper curve. If, in this typically recruiting lesion, recruitment were to depend upon the activity of the hair cells in the form of cochlear potentials, the curves should unite at higher intensities instead of showing the present nonrecruiting slope. The results of Davis and his associates[11] suggest, on the other hand, that the maximum response is lowered less than the threshold, and it might be said to represent partial recruitment.

At the present state of our knowledge I suggest that the nerve endings around the hair cell bases may possibly be important also in the origin of recruitment, although the idea that the mechanical movements of the basilar membrane might directly stimulate the nerve endings, is not consistent with the present belief; however, many of Saxén's presbycusis case records indicate good hearing even in cases where the hair cells had suffered intensive damage but the nerve endings were intact. Further data on this question are required.

Indirect Recruitment Tests.

A number of other tests have been devised to take the place of loudness balance tests, or some have been claimed to be independently of great clinical significance. In this connection I can discuss only briefly some of these tests. For a more complete evaluation the reader is referred to an earlier paper.[21]

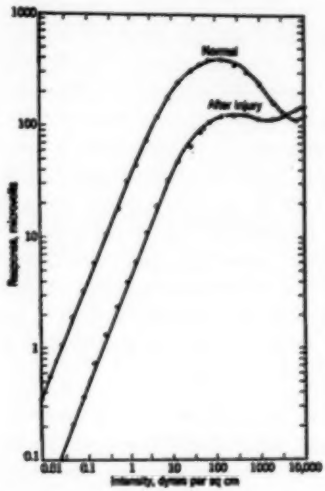


Fig. 2. Intensity functions for the cochlear responses of the cat on stimulation with a 1000 c.p.s. tone, before and after injury by overstimulation with this tone. Reproduced from "Physiological Acoustics," p. 295, by Wever and Laurence.[20]

Together with Hirsh and Goodman,[22] I have shown that the tests for intensity difference limen do not differentiate cochlear from retrocochlear lesions: the figures for IDL are of the same magnitude for both groups. Our testing was done by the method of comparing the intensity of two separate tones, but there is also enough evidence to testify that the use of modulated tones[23,24] does not take us further in the differential diagnosis.

The same can be applied to tests for frequency difference

limen. Some authors[25,26] claim that especially an increased FDL signifies recruiting deafness and cochlear lesions; others say that this very finding is frequent in retrocochlear pathology.[27] It is difficult to see how the size of the FDL could be different in these two main categories when one remembers that diplacusis, a change in pitch perception, occurs in cochlear as well as in retrocochlear lesions.

Tests for either per- or post-stimulatory fatigue[28-31] have been recommended for differential diagnosis; however, control experiments have shown that abnormal results appear in the two main groups of perceptive deafness, and a large proportion of the cases show results comparable to normal.[32-36] We may take as an example continuous threshold recording at a fixed frequency which exemplifies a special kind of per-stimulatory fatigue. Hood[28] has claimed that abnormal fatigability in this test is typical of an end-organ lesion, and Reger and Kos,[36] and Kos[37] have reported cases of acoustic tumors with highly fatigable ears at threshold stimulation. In my experience[35] a good many cases in both groups show only minor fatigability during a four minute test, but there are cases in both the cochlear and retrocochlear group that show also abnormally high fatigue.

The width of the excursions in a self-recording audiometer has also been used as a localizing test in audiometry.[38] It seems true that very small excursions, indicating rapid growth in audibility, are a common phenomenon in cases with asymptotic recruitment at higher frequencies. At the lower tones, below 1000 c.p.s., and in the groups of straight or delayed recruitment, the size of the excursions is generally larger and allows no conclusions as to the recruitment phenomenon.[35]

Masked audiograms, either with white noise[39] or with saw tooth noise,[40] have also been advocated in the differential diagnosis. In control measurements with critically controlled stimuli it has been shown that noise audiometry is not helpful for this purpose.[41,42]

Speech Tests.

During the last decade it has been established beyond doubt

that the recruitment phenomenon is accompanied with poor discrimination for speech. This has been reported among others by Dix, Hallpike and Hood,[43] by Eby and Williams,[44] by Huizing and Reyntjes,[45] and in our own series.[22,46] Speech function depends mainly upon two facts, *viz.* the characteristics of the language and the threshold loss for speech. Using the Finnish language, which is rich in vowels, the ability to discriminate is not greatly reduced until the threshold

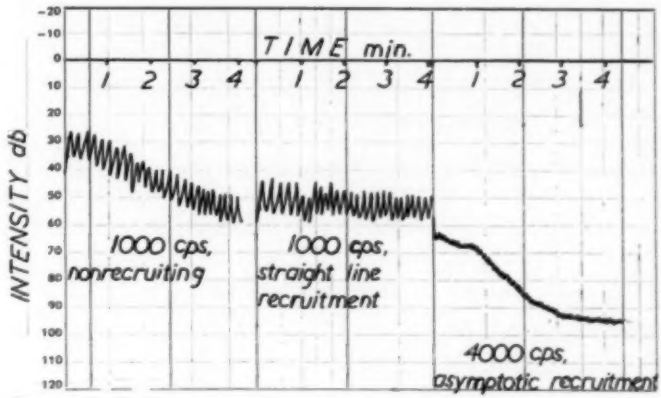


Fig. 3. Continuous 4 min. threshold recordings in various forms of perceptive deafness. Temporary threshold shift may appear in recruiting as well as in nonrecruiting deafness. The width of the tracings may remain large also in recruiting deafness, especially at lower frequencies.

loss exceeds 40 db[46] but using the American PB test this already occurs in cases of 20 db loss.[22]

It was believed at first that in retrocochlear deafness, even when it is severe, speech discrimination is better than in end-organ lesions. This was the experience of Dix, Hallpike and Hood[43] and of Eby and Williams[44] in acoustic neurinomas; and at least in the latter series, there were enough data of masking to convince the reader that shadow hearing was not present. The ability to discriminate speech was reasonably good and increased with increasing amplification; however, increasing clinical experience brought out deviating results.

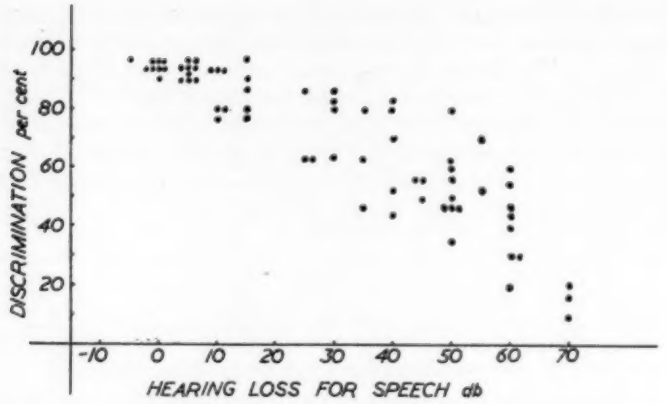


Fig. 4. The relation between discrimination ability and hearing loss for speech in 69 cases with complete recruitment, tested with the Finnish speech test material.[46] Discrimination suffers notably when the threshold loss exceeds 40 db.

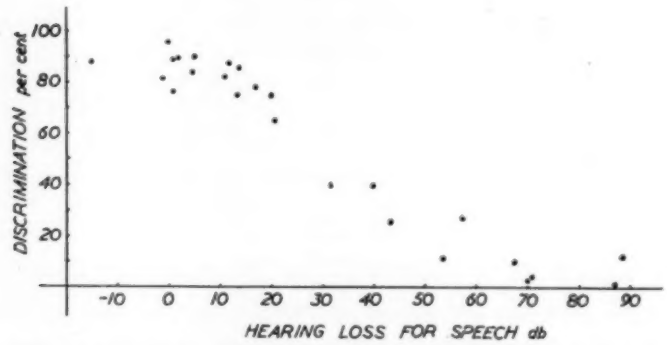


Fig. 5. The relation between discrimination ability and hearing loss for speech in 27 cases with complete recruitment, tested with the American PB material. Discrimination suffers greatly already after 20 db threshold loss. Data from Hirsh, Palva and Goodman.[22]

Poor speech discrimination in acoustic neurinomas has been reported in seven cases by Liden,[47] in two by Schuknecht and Woellner,[48] and in one by Harbert and Sataloff.[49] The two cases of our own,[22] to which Walsh and Goodman[50] later added one, also showed very poor articulation function.

In quite small threshold losses, discrimination for speech can be disproportionately poor or even nil.

The testing experience at our laboratory still suggests that in nonrecruiting neural pathology, excluding acoustic tumors, speech function is lowered but reasonably good. It may be that the presence of an acoustic tumor in a nerve reduces the nerve fibers to such an extent that delivery of all complex signals becomes impossible.

In conclusion, we do not at present have such audiological tests as would tell us that in one case there is a pure cochlear lesion and in another a pure retrocochlear lesion with 100 per cent certainty. We should, therefore, avoid depending too much on audiological tests alone. Audiometric tests are an extremely important asset in case evaluation, but it must be remembered that final diagnosis can be made and the lesion located only on the basis of complete anamnestic and clinical data, supported by the results of auditory tests.

REFERENCES.

1. Palva, T., and Ojala, L.: Middle Ear Conduction Deafness and Bone Conduction. *Acta Otolaryngol.*, 45:137, 1955.

2. Fowler, E. P.: A Method for the Early Detection of Otosclerosis. *Arch. Otolaryngol.*, 24:731, 1936.

3. Dix, M. R.; Hallpike, C. S., and Hood, J. D.: Observations upon the Loudness Recruitment Phenomenon with Especial Reference to the Differential Diagnosis of Disorders of the Internal Ear and the VIIIth Nerve. *Jour. Laryngol. and Otol.*, 62:671, 1948.

4. Hallpike, C. S., and Cairns, H.: Observations on the Pathology of Ménière's Syndrome. *Proc. Roy. Soc. Med.*, 31:1317, 1938.

5. Dix, M. R., and Hallpike, C. S.: The Pathology, Symptomatology and Diagnosis of Certain Common Disorders of the Vestibular System. *Ann. Otol., Rhinol. and Laryngol.*, 61:986, 1952.

6. Altmann, F., and Fowler, E. P., Jr.: Histological Findings in Ménière's Symptom Complex. *Ann. Otol., Rhinol. and Laryngol.*, 52:52, 1943.

7. Lindsay, J. R.: Ménière's Disease. Histopathologic Observations. *Arch. Otolaryngol.*, 39:313, 1944.

8. Lindsay, J. R.: Labyrinthine Dropsy. The Laryngoscope, 56:325, 1946.

9. Smith, K. R.: The Problem of Stimulation Deafness. *Jour. Exp. Psychol.*, 37:304, 1947.

10. Wever, E. G., and Smith, K. R.: The Problem of Stimulation Deafness. *Jour. Exp. Psychol.*, 39:238, 1949.

11. Davis, H., et al.: Acoustic Trauma in the Guinea Pig. Jour. *Acoust. Soc. Amer.*, 25:1180, 1953.

12. Rüedi, L.: Occupational Injuries of the Ear. *Acta Otolaryngol.*, 41:118, 1952.

13. Davis, H., et al.: Temporary Deafness Following Exposure to Loud Tones and Noise. *Acta Otolaryngol.*, Suppl. 88, 1950.

14. Palva, T.: Occupational Deafness in Telephone Exchange Workers. *Acta Otolaryngol.*, 47:510, 1957.

15. Saxén, A.: Pathologische Anatomie und Klinik der degenerativen Erkrankungen des Gehörorgans. *Erg. d. Pathol.*, Bd. 34, 1938.

16. Palva, T.: Audiometric Diagnosis of Hearing Defects. *Suom. Lääk. l.*, 12: 452, 1956.

17. Pestalozza, G., and Shore, I.: Clinical Evaluation of Presbycusis on the Basis of Different Tests of Auditory Function. The Laryngoscope, 65:1136, 1955.

18. Dix, M. R., and Hood, J. D.: Modern Developments in Pure Tone Audiometry and Their Application to the Clinical Diagnosis of End-Organ Deafness. *Jour. Laryngol. and Otol.*, 67:343, 1953.

19. Fowler, E. P.: The Recruitment of Loudness. The Laryngoscope, 60:680, 1950.

20. Wever, E. G., and Laurence, M.: "Physiological Acoustics." Princeton University Press, Princeton, N. J., 1954.

21. Palva, T.: On Recruitment Testing. *Arch. Otolaryngol.*, 66:93, 1957.

22. Hirsh, I. J.; Palva, T., and Goodman, A.: Difference Limen and Recruitment. *Arch. Otolaryngol.*, 60:525, 1954.

23. Lund-Iversen, L.: An Investigation on the Difference Limen Determined by the Method of Lüscher and Zwislocki in Normal Hearing and in Various Forms of Deafness. *Acta Otolaryngol.*, 42:219, 1952.

24. Fournier, J. E.: Le Seuil Differentiel de la Hauteur. *Proc. Vth. Int. Cong. Oto-Rhinol-Laryngol.*, p. 242, 1953.

25. Schubert, K.: Diagnostische Fortschritte durch Tonhöhenunterschiedsschwellenmessung. *Arch. Ohrenheilk*, 161:499, 1952.

26. Meurman, O. H.: The Difference Limen of Frequency in Tests of Auditory Function. *Acta Otolaryngol.*, Suppl. 118:144, 1954.

27. Azzi, A.: The Sensibility of the Human Ear to Frequency Modulation. *Proc. 1st. Int. Cong. Audiol.*, p. 57, Leyden, 1953.

28. Hood, J. D.: Auditory Fatigue and Adaptation in the Differential Diagnosis of End-Organ Disease. *Ann. Otol., Rhinol. and Laryngol.*, 64:507, 1955.

29. Hallpike, C. S., and Hood, J. D.: Some Recent Work on Auditory Adaptation and Its Relationship to the Recruitment Phenomenon. *Jour. Acoust. Soc. Amer.*, 23:270, 1951.

30. Hood, J. D.: Studies in Auditory Fatigue and Adaptation. *Acta Otolaryngol.*, Suppl. 92, 1950.

31. Huizing, H. C.: The Relation Between Auditory Fatigue and Recruitment. *Acta Otolaryngol.*, Suppl. 78:169, 1948.

32. Palva, T.: Studies of Per-stimulatory Adaptation in Various Groups of Deafness. The Laryngoscope, 65:829, 1955.

33. Lierle, D. M., and Reger, S. N.: Experimentally Induced Temporary

Threshold Shifts in Ears with Impaired Hearing. *Ann. Otol., Rhinol. and* Laryngol., 64:263, 1955.

34. PALVA, T.: Post-stimulatory Fatigue in Diagnosis. *Arch. Otolaryngol.*, (In Press).

35. PALVA, T.: Self-recording Threshold Audiometry and Recruitment. *Arch. Otolaryngol.*, 65:522, 1957.

36. REGER, S. N., and KOS, C. M.: Clinical Measurements and Implication of Recruitment. *Ann. Otol., Rhinol. and Laryngol.*, 61:810, 1952.

37. KOS, C. M.: Auditory Function as Related to the Complaint of Dizziness. THE LARYNGOSCOPE,, 65:711, 1955.

38. LUNDBORG, T.: Diagnostic Problems Concerning Acoustic Tumors. *Acta Otolaryngol.*, Suppl. 99, 1952.

39. LANGENBECK, B.: "Leitfaden der praktischen Audiometrie." Georg Thieme Verlag, Stuttgart, 1952.

40. BOCCA, E.: Interrupted Masking as a Differential Test of Auditory Function. *Acta Otolaryngol.*, 34:452, 1951.

41. PALVA, T.; GOODMAN, A., and HIRSH, I. J.: Critical Eveluation of Noise Audiometry. THE LARYNGOSCOPE, 63:842, 1953.

42. PALVA, T.: Studies of Hearing for Pure Tones and Speech in Noise. *Acta Otolaryngol.*, 45:231, 1955.

43. DIX, M. R.; HALLPIKE, C. S., and HOOD, J. D.: "Nerve" Deafness: Its Clinical Criteria, Old and New. *Proc. Roy. Soc. Med.*, 42:527, 1949.

44. EBY, G., and WILLIAMS, H.: Recruitment of Loudness in the Differential Diagnosis of End-Organ and Nerve Fiber Deafness. THE LARYNGOSCOPE, 61:400, 1951.

45. HUIZING, H. C., and REYNTJES, A. J.: Recruitment and Speech Discrimination Loss. THE LARYNGOSCOPE, 62:521, 1952.

46. PALVA, T.: Finnish Speech Audiometry. *Acta Otolaryngol.*, Suppl. 101, 1952.

47. LIDEN, G.: Speech Audiometry. *Acta Otolaryngol.*, Suppl. 114, 1954.

48. SCHUKNECHT, H. F., and WOELLNER, R. C.: An Experimental and Clinical Study of Deafness from Lesions of the Cochlear Nerve. *Jour. Laryngol. and Otol.*, 69:75, 1955.

49. HARBERT, F., and SATALOFF, J.: Clinical Application of Recruitment and Masking. THE LARYNGOSCOPE, 65:113, 1955.

50. WALSH, T. E., and GOODMAN, A.: Speech Discrimination in Central Auditory Lesions. THE LARYNGOSCOPE, 65:1, 1955.

DR. HIRSH: Are there any comments on Dr. Palva's paper?

DR. JOSEF ZWISLOCKI, (Cambridge, Massachusetts): I have comments on the two papers of Dr. Palva and Dr. Carhart.

I am not quite sure when it is safe to say that the difference between bone conduction and air conduction differentiates conductive from perceptive deafness. There is one thing which is established beyond a reasonable doubt today, *i.e.*,

that the conduction of sound in the inner ear is essentially the same for air and bone conduction.

DR. WILLIAM G. HARDY, (Baltimore, Maryland): There are gap areas in the diagnosis of hearing losses that need much work. For example, we cannot offer much of value to the patient when we find nerve degeneration or an advanced case of otosclerosis. There is a toxic condition that leads to nerve degeneration, but we do not know just what it is.

In another area is the case of deafness caused by a single blow to the head. We hear much about acoustic trauma, and shock pulses causing total deafness, but clinically, we are frequently presented with the case of a man who receives a blow to the head which is not severe enough to knock him down, yet he loses his hearing. Or again, he may be knocked down, but not hard enough to cause a fracture of the skull or produce unconsciousness, but still he has a total loss of hearing. I think something like this may be the result of a hemorrhage into the labyrinth.

In still another area, the inner ear, we see the effects of virus disease. Measles and mumps may disturb the hearing, even without an open infection. A virus may attack and the lesion may be entirely in the endolymphatic system and begin, for example, in the stria vascularis and the tectorial membrane may roll up. We get a greater destruction of the stria on the basal turn, in these cases, and the stria may even disappear at that point.

We have many cases of deafness which we cannot explain, some of which are connected with ocular disturbances. This may be a toxic involvement through the round window.

Then there are the intriguing diseases, such as Ménière's disease, where there is a shift in threshold and the hearing is depressed more in the low tones. Why is there only a low tone deafness? In theory this is only possible on the basis of an inner ear conductive loss, but the presence of recruitment makes this not very probable; however, the shift from a high to a normal hearing threshold may take place in only a few hours. This again favors the theory of the condition of the hair cells themselves as being important.

I have patients who have presbycusis *before* they developed Ménière's disease. Deafness seems to have been caused by a marked disturbance of the wall of the vestibule.

CLINICAL ASPECTS OF CORTICAL DEAFNESS.

ETTORE BOCCA,

University of Milan,

Milan, Italy.

The clinical examination of the peripheral organ of hearing has undergone a radical evolution in the recent decades, thanks to electrophysiological investigation of the cochlea, and to the introduction of the audiometer; however, it has not had its counterpart in a better knowledge of the auditory experience as a whole.

More and more attention has been devoted to audiometry, with the result that we know a lot more about hearing but as little as ever about listening. Since the assertion of Collet in 1920, that we know nothing about the cortical troubles of hearing, things do not seem to have improved much until 1953, when Aboulker stated that the semeiology of the cortical disorders of hearing is still in its infancy. Two years later Alajouanine, in his beautiful book on the functions of the temporal lobe, concluded that it is so far impossible to make an audiometrical diagnosis of a lesion of the auditory cortex.

As far as we are dealing with the organ of hearing, physiological, physiopathological and clinical investigation may avail themselves of the same stimuli: pure tones, or simple sounds. Before these stimuli, the organ of hearing behaves in a well-defined fashion, according to well-known physical and mathematical laws.

The appreciation of loudness casts a shadow upon the value of some physical determinations, but that problem, too, has had a satisfactory explanation on a psychophysiological basis. As far as tones and simple sounds are concerned, the response

in the experimental animal may be compared with the responses which are obtainable in the clinical experiment in man; however, things get more and more complicated as we consider the analysis and the identification of a meaningful message. The electrical response of single cortical areas to pure tones, or clicks, provides an excellent basis for the knowledge of the limits, and the excitatory processes, of the auditory cortex, but it does not help in explaining how the cortex may elaborate the psychological aspects of a verbal message.

The conditioned thresholds to sounds in the decorticated animal only prove that most acoustically conditioned reflexes take place at some level below the cortex. The demonstration afforded by that animal, previously conditioned to different sequences of stimuli, which fails to give differentiated responses after its cortex has been removed, is the only experimental hint to the importance of the cortex in cortical discrimination of auditory patterns. Recently, further observations upon this point have been reported. The function of the auditory cortex must not be regarded as analytical; on the contrary, it is mainly synthetical. The different attributions of sounds must be faithfully transmitted from the ear to the centers in order that they may be correctly recognized, but, when the sound is a meaningful verbal message, it is also necessary that they reach the centers with a certain order in space and time. "Reversed" words are not comprehensible, in spite of the fact that they contain exactly the same frequencies and intensities; a poorly known foreign language is not understood if it is spoken rapidly. These few facts may serve as an example that there is a wide difference between the recognition of the physical qualities of a sound and the process of identification of a verbal pattern. The former depends upon the working of the auditory apparatus as a whole; the latter is a specific function of the auditory cortex. This is a very obvious and widely accepted fact, which should have long since orientated all clinical investigations of hearing at a cortical level.

Otologists of many countries have been busy in recent years trying to reveal cortical disturbances of hearing by means

of pure tones, which are to be considered as the specific stimulation of the cochlea, cochlear nerve, and the first stations of the auditory pathway. The scanty data available in the literature are equal only to the scarce importance of the observations.

If we devote some attention to the works of all the great neurologists and psychologists of the last 70 years this seems to be rather surprising. In the chapters dedicated to the problem of aphasia, the way to further research in the field of cortical deafness is clearly indicated. Until the physiological experiments are able to bridge the gap between form and experience (which is highly improbable) we shall be obliged, when studying sensation at a psychological level, to set apart all the traditional experimental and clinical methods, and to avail ourselves of testing material of a purely psychological value. Pure tones and simple sounds must be abandoned, and words or sentences must be resorted to; because they alone own the symbolic, emotive, mnemonic, and evocative qualities which acquire their significance at a cortical level.

As a matter of fact, after the failure of tonal audiometry, hearing for the voice has been tested by some authors, in cases of pathology of the temporal lobe; the results did not significantly differ from normal, so that any further possibility of audiometric investigation of the auditory cortex seemed to be precluded. At the present state of knowledge there is a sharp passage, in the temporal lobe pathology, from normal hearing to word-deafness, which represents the final stage of most severe lesions. We have no means of revealing audiometrically the early or pre-aphasic stages.

If we bear in mind the importance of quality, space, time, and rhythm as factors governing the cortical identification of a verbal pattern, we may be easily persuaded that modern electroacoustic engineering gives us a chance of altering one or more of these factors in a way which can be exactly defined and easily repeated; an entirely new testing material may be put at our disposal, for the detection of early troubles of cortical hearing, in the same way as the audiometer reveals early and unsuspected troubles of the frequency and intensity

function. It is not difficult to explain why, in limited lesions of the temporal lobe, evident disorders of verbal hearing are rare events.

The nervous sensory apparatus works according to a principle of redundancy, which is very common to all natural organizations. As a consequence, even if a fairly large number of its structures are put out of function, sufficient contacts are always possible between the centers and the external world.

On the other hand, speech also, as a means of communication, works along the same principle. Every speech sound contains an enormous amount of information units, far outnumbering the minimum that is theoretically required for its differentiation from another sound; as a consequence, even if a certain number of those units get lost, due to faulty transmission or reception, the speech sound is still recognizable; moreover, if one considers the rather rigid way in which speech sounds are combined into a word, or words into a sentence, a very considerable loss of quality is necessary to make speech no longer intelligible. It appears logical to suppose that the conditions for the identification of a verbal message grow more difficult if the poor quality of the message is associated with a poor working of the neural sensory structures; in other words, when a peripheral distortion is associated to what we may call a central distortion. The distortion may affect intensity, frequency, and time. Any alteration of these factors will alter the value of the equation

$$I \text{ (amount of information)} = 2\ t\ w \log \frac{S + N}{N}$$

(Where t is the duration of the signal, w is the width of the usable frequency range, S the maximum amplitude of the signal, N the minimum discernible difference of intensity).

thereby reducing or increasing the amount of information.

Other factors such as space and rhythm must also be taken into consideration; therefore, we developed a series of clinical audiometric investigations whose logical basis was that of sensibilizing the traditional speech tests by making them more difficult in several ways (cutting of frequencies, accel-

eration, periodic interruption, increase of the length of the message, alteration of meaning, rhythm, accentuation). Some of these tests have already been performed in an extensive number of cases of tumors of the temporal lobe, and of degenerative changes of the cerebral cortex, due to old age. In all these cases traditional tonal and speech audiometry failed to reveal any significant deviation from normal. Some other tests have been so far experimented with in a more limited number of cases, mainly or exclusively old-aged subjects.

FACTORS AFFECTING THE CORTICAL RECOGNITION OF THE VERBAL IMAGES:

Factors	Related Tests
Physical Qualities of the Verbal Pattern	Distorted Voice; Interrupted Voice.
Duration of the Signal	Accelerated Voice.
Length of the Message	Long Sentences or Lists of Words.
Meaning	Meaningless Sentences; "Reversed" Sentences: (Ex.: the cat is on the table; table the on is cat the).
Rhythm	Prosodic Alteration (Accent Shift); Aperiodic Variations of Speed; Reversed Sentences.

Fig. 1.

1. Distorted voice: Lists of PB disyllabic words have been presented monaurally at 50 db above threshold, through a low-pass filter capable of reducing discrimination to 70 to 80 per cent in the normal subject. Temporal lobe tumors present an average discrimination which is poorer in the ear contralateral to the tumor. (50 per cent vs. 65 to 75 per cent in the ipsilateral ear.)

2. Interrupted voice: A discrimination of 80 per cent of PB disyllabic lists is the rule in the normal subject, if the voice at 50 db above threshold is interrupted periodically 10 times per second, the ratio voice:silence being 1:1. Tumors of the temporal lobe show worse discrimination in the ear contralateral to the lesion (30 to 40 per cent less as an average).

3. Accelerated voice: If the number of words per minute

of the speech is increased from the normal (about 150 words per minute) to about 350 words per minute, the discrimination always approaches 100 per cent in the normal subject; however, the threshold is raised by 10 to 15 db. Tumors of the temporal lobe may present a normal threshold shift, but the discrimination never attains 100 per cent in the contralateral ear.

4. Long messages: When listening at a comfortable level, a normal subject is able to repeat 100 per cent of obvious

AGED PERSONS.

Test		Response
Distorted Voice	+	Fairly Good.
Interrupted Voice	+++	Very Poor in Both Ears.
Accelerated Voice	+++	
Long Sentences or	+++	Very Poor in Both Ears.
Long Lists of Words	++	
Meaningless Sentences	+	Fairly Good.
Reversed Sentences	+++	Extremely Poor.
Prosodic Alteration	+++	Extremely Poor.
Velocity Fluctuations	++	Poor.

TEMPORAL LOBE TUMORS.

Test		Response
Distorted Voice	+++	Poor in Contralateral Ear.
Interrupted Voice	+	
Accelerated Voice	++	
Long Sentences or	+++	Poor in Both Ears.
Long Lists of Words	++	Only in Left Side Lesions. (Right-handed Pts.)

(+ indicates validity of the test.)

Fig. 2.

sentences of ten words, and respectively 70 per cent of 15 words and 40 per cent of 20 words. Tumors of the temporal lobe show a very poor performance (respectively 70-10-0 per cent for monaural left and right, or binaural audition, when the tumor is located in the left hemisphere in right-handed patients. No deviation from normal is observed in right-sided tumors.

The results obtained appear to be specifically indicative of an impairment of the auditory cortex, as no similar re-

sponses have been observed in the many cases where the tumor was located outside the temporal lobe.

It is interesting to note that, as far at least as the integration of a distorted message is concerned, there seems to be no real dominance of one hemisphere over the other. The poorer response is always in the contralateral ear, regardless of the side where the lesion is located; however, for the function of memory, the left hemisphere in right-handed patients is clearly dominant. Cases of severely increased intracranial pressure often give bilaterally poor responses.

The observations made in tumoral pathology prompted us to investigate the response to the same tests in old-age subjects, showing the best possible audiometric curves in relation to age and the best possible performance in psychometric tests, and normal responses to traditional speech audiometry. The results have been even more demonstrative than those observed in the group of temporal lobe tumors; of course, no difference existed in monaural tests between the response of the two ears. The only exception was observed in the test of distorted voice. Discrimination was better in aged subjects than in temporal lobe tumors. This may be tentatively explained by the fact, that due to the progressive loss over high frequencies, the aged persons have been slowly getting accustomed to the cortical integration of messages lacking the higher portion of their acoustic spectrums.

The interrupted voice test gave much poorer responses than in tumor cases. The same happened with the accelerated voice test; the long sentences test gave as poor results. Some of the poorer responses may depend upon the greater synaptic delay in the cortex. In other cases, they may be due to the lack of partial compensation by the contralateral temporal lobe. The comparative evaluation of the results of our tests in the old age and tumor group of cases is an implicit confirmation that the auditory troubles of old age are mainly cortical in origin, and that insofar as the process of cortical elaboration of the message is concerned, it does not matter whether the cortical lesions are brought about by compression and invasion, or by primary atrophic and degenerative changes.

This fact has persuaded us in recent months to extend further our psychoacoustic researches in the aged subjects, who form a far more numerous and homogeneous group than tumor cases, with the conviction that the results may be a proof of the validity of new testing material, and an indication of its possible employment in the study of cortical pathology from other causes.

The new testing material used consists of:

1. Meaningless sentences: The importance of meaning does not much affect the correct repetition of sentences, with results only 20 per cent less as compared with the control group.

2. Alteration of rhythm: The importance of rhythm seems to be of paramount importance in the correct recognition and repetition of sentences. Rhythm may be altered by, *1.* irregularly varying the output velocity of the speech, or *2.* shifting the accent of words to wrong places.

In the old-age group, when compared with the control group, the percent of correct responses falls respectively from 100 to 70 per cent, and from 50 to 0 per cent.

These investigations further helped us to establish which functions of the auditory cortex and associated areas are more liable to be compromised, and which tests are more suitable for the detection of a cortical impairment of hearing, whose clinical evidence is a very late and very rare occurence. Modern electroacoustic facilities allow us to multiply these tests, and to perform them under conditions of so far unknown reliability. A first step seems to have been made in the creation of a new semeiology of the cortical disturbances of hearing.

This new semeiology is not only diagnostically important, but it entails interesting physiological and psychological implications, *viz.* the relative importance of crossed and uncrossed fibers along the auditory pathway, and the dominance of one hemisphere over the other; moreover it leads to a considerable widening of the term of "deafness' as it is generally accepted today, especially in old age, and offers the

hope of a better solution of some of the many problems regarding prothesic adaptation and the re-education of the deaf.

DR. HIRSH: Are there any questions or comments on Dr. Bocca's paper? Dr. De Sá has a comment.

AUDIOLOGIC FINDINGS IN CENTRAL NERVE DEAFNESS.

GERALDO DE SA,

University of Recife, Brazil.

The detection of nerve deafness of central origin, is the latest trend in audiology. The development of electric audiometry, and mainly of speech tests, brought a greater interest of the audiologist and the otologist in general to the possibility of the existence of a complex pathology, under the uncertain diagnostic of nerve deafness. For the past five years literature has been abundant on the subject of deafness caused by tumors and traumatism; therefore, greater interest arose with the better knowledge of the possibilities of speech testing. Attention is being called to the importance of speech discrimination losses for the detection of central nerve deafness since 1953 by Theo. E. Walsh. Also, a better study of the recruitment phenomenon permitted the audiologist to separate cases of cochlear and post-cochlear nerve deafness.

Perceptive aphasia was studied for a long time by the neurologist, and only recently was developed in the field of the otologist. It seems to us that the two portions of the acoustic nerve; terminal organ, and acoustic pathway and brain projections, should be studied separately, being considered the cochlea as a sound selector and the pre-cortical and cortical areas as the final analyzers. The audiometry with pure sounds would be, evidently, of great importance for the diagnosis of diseases of the selective organ, the organ of Corti; however, the identification of the composed sounds, and more especially of the speech sounds, being accomplished on the pre-cortical or cortical centers, would be better made

with the help of testing measures that demand more intensive cerebral implication.

This seems to be today, a generalized idea for the audiologists. To test the perception of a word, no better testing material could be available than the word itself. For all of us dedicated to the speech audiometry the importance of the discrimination tests for diagnosis soon become apparent. Besides, the use of phonetically balanced word lists holding identical percentage of sounds of a language, would permit testing a patient unable to hear this language well, with material adequate for his disability.

In our country, where otosclerosis affects only 15 per cent of the cases of deafness, the most common symptom brought by a patient to the otologist is to hear, but not to understand. It seems to be that nerve deafness is our real toll. As a matter of fact in nearly 600 cases of deafness studied in the last four years, we found about 40 per cent of cases of so-called "nerve deafness." Under these local circumstances the discrimination tests became of great importance to us.

We used on our patients, for determination of discrimination score, phonetically balanced lists, prepared by ourselves at Central Institute for the Deaf, St. Louis, Mo., in June, 1952. Since the beginning of the work with the phonetically balanced word lists, in September, 1952, we decided to observe a specific approach, by registering each answer of the patient, and not only establishing a curve. In this way we believed we could, after enough experience, see which sounds were most commonly lost by the patient in the several diagnostic groups.

Employing phonetically balanced lists, containing all the sounds of the language in identical percentage, it was easy to disintegrate each monosyllable lost, in its phonetical components, and to establish definite medias referring to all the phonems of the language. This study is presented today, and might lead to interesting conclusions. On the other hand a careful control of the improvement of hearing and discrimination after therapeutical measures could be established.

Based on our observations, we agreed with Dr. Walsh that the cases of otosclerosis showed very little discrimination loss,

while the nerve deafness showed a greater one, and that a certain type of deafness showed an extremely great discrimination loss. In cases of nerve deafness, some showed recruitment and some did not. Those with recruitment, we named cochlear deafness, and on our charts are represented by five cases of hydropsy; three cases of toxic neuritis, and two cases of acoustic trauma.

Among the cases with no recruitment, that we named retro-cochlear deafness, we charted two cases of skull trauma; two cases of epilepsy with focus on the left temporal lobe according to electroencephalography findings; one case of multiple sclerosis; one case of general toxicosis; two cases of supposed cortical deafness, and two cases of presbycusis of the Schuknecht physiological type.

Among the cases of otosclerosis, ten patients with no signs of nerve involvement were chosen. Their media of pure tone loss was 65 db, and their media of discrimination was 84 per cent. Among the 48 phonems in the Portuguese language, 15 were heard by all the patients; 1160 phonems were given to the ten patients, and only 83 were lost, giving a media of 7.15 per cent of discrimination loss. Among the phonems lost the F's came with the highest percentage, 40 per cent, followed by the S's with 35.5 per cent. The D's, the B's and the AN's followed with a smaller percentage, and among the rest, the losses were very small.

The cases of recruiting nerve-deafness showed a very different aspect. Their media of pure tone loss was 40 dbs, and their discrimination was 70 per cent. Five cases had pure tone around 20 to 25 c.p.s. Among the 48 phonems in the Portuguese language (as spoken in Brazil) 11 were heard by all the patients. From the given 1160 phonems 145 were lost, giving a media of 12.5 per cent of discrimination loss. The F's were the phonems lost more often, 70 per cent; and the S's gave a percentage of loss of 63.328 per cent; the IN's and the B's followed with 40 per cent and of the diphthong AI 50 per cent was lost. Of the D's 30 per cent were lost, and the Z's, UN's, V's and T's followed. The rest of the phonems were in a smaller percentage. It was evident that cases of nerve deafness with recruitment, lose more consonants,

Otosclerosis

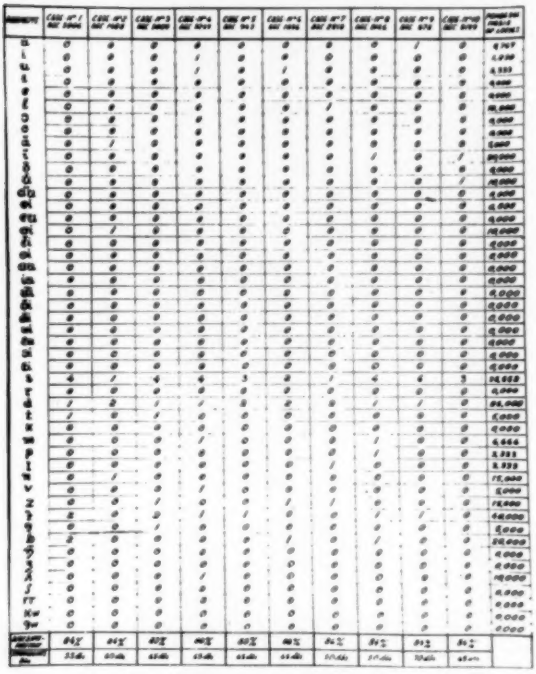


Fig. 1.

among them, F's, the S's, the D's, the T's and the B's. One could detect the diagnosis by the almost complete losses in S's, that were scarcely heard at all. Those cases showed also sloping curves towards the high frequency tones from 2000 c.p.s. up. Their bone conduction was poor, but present.

Now we come to the cases that are mainly the reason for this communication—the nerve deafness cases without recruitment, that we charted as retro-cochlear deafness. Their media of pure-tone loss was 45 dbs., and one case had no pure tone loss at all. Their media of discrimination was 45 per cent. Among the 48 phonems in the Portuguese language,

Cochlear Deafness
(ACCLIMITMENT)

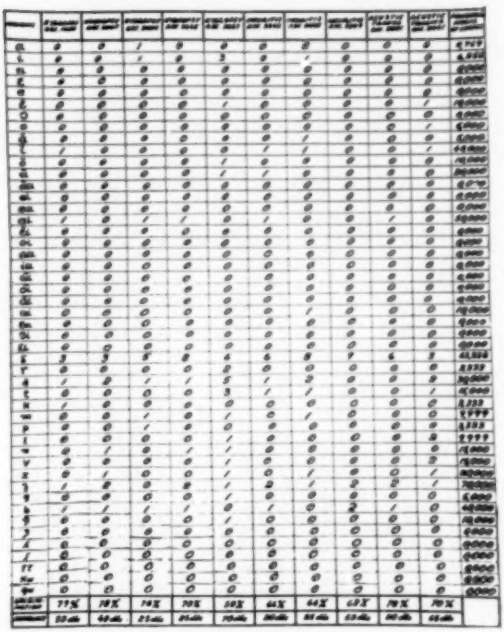


Fig. 2.

none escaped being lost by some patients. From the given 1160 phonems, 347 were not heard, which gives a media of 29.913 per cent of discrimination loss.

On the selection of the phonems lost, we saw a complete change from the other types of deafness. The AN's, that until now showed practically no losses, showed 90 per cent loss. The F's presented 70 per cent of losses; the IN's and the UN's showed 60 per cent, and so did NN; also a nasal sound. The B's showed 65 per cent loss, and again the nasal sounds ON and the nasal diphthong AO showed 50 per cent

Retro-Cochlear Deafness
(NO RECRUITMENT)

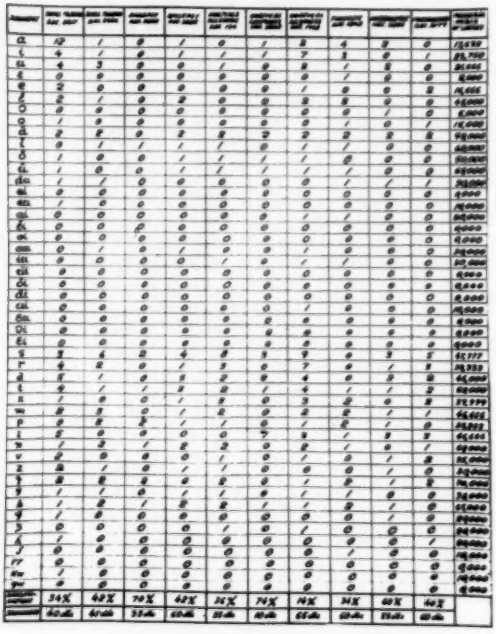


Fig. 3.

loss, while another nasal phonem, the EN, showed 45 per cent. The T's lost 50 per cent, and the B's 65 per cent; the S's 47 per cent; the M's 46 per cent, and the L, an easy phonem in Portuguese, 46 per cent loss. The K's, V's, Z's and G's all showed losses up to 30 per cent.

It was interesting to observe the discrimination of phonems lost by other types of patients, and the appearance of fantastic answers, difficult to find in other types. In the ten patients, more than 50 fantastic answers were given, which raises the percentage to more than 10 per cent. Although showing heavy loss, the S's were relatively well heard

compared with other types of deafness. Mistakes among consonants were very common. The most frequent were: F or S and T; T for D and D for T; P for T, and D and F for S. Among the cases, we have charted two skull traumas without any other disablement than dizziness and deafness. Case No. 2986 does not respond to any caloric excitement in either ear; the two cases of epilepsy showed abnormal encephalogram with focus of slow activity in the left temporal area with sharp and spiky waves on the left anterior temporal zone. Case No. 2892 showed, in the Cawthorne-Hallpike test, directional preponderance for the left. Examined thoroughly by the neurologist, Dr. M. C. Barros, no other nervous symptom was found. It would be interesting to note that another patient with focal epilepsy on the right side showed no loss of discrimination worthwhile; pure-tone media around 40 dbs. and discrimination score 90 per cent.

One case of multiple sclerosis had a linear, flat curve around 55 dbs; no bone conduction, and a very poor discrimination. It has been followed now for six years and hears nothing at all for the last two years. Regarding the general toxicosis, one patient has for a long time taken alcoholic drinks to excess and has a great difference between the left and right ears with very bad discrimination in both. Hearing in one ear is today almost lost, and the other almost as bad, as he continues the same habit.

Other cases of supposed cortical deafness refer to a patient with almost normal pure-tone level, and great discrimination loss, and another with great loss of discrimination and pure tones, that recurred with clinical treatment to a 90 per cent discrimination score, this patient being long underfed because of religious motives. The two cases of presbycusis showed no recruitment and no history of toxidity and could be put in the class of general senile breakdown.

We could not find in any examination signals that could be referred to lesions on the pathway between the cochlea and the cortex, but we are convinced that patients with flat pure tone threshold, great discrimination loss, mainly of nasal sounds, many consonant mistakes, frequent senseless answers, no recruitment, bone conduction very low or absent, have

retro-cochlear deafness and if all these signals are backed by the existence of directional preponderance or electroencephalographic findings, cortical deafness could be suspected.

SUMMARY.

The author presents several cases of central deafness, some of them confirmed electro-encephalographically, and studies their audiological findings:

a. Tono-audiograms are presented for each case, emphasis being placed on the linear aspect of the curves.

b. Portuguese speech tests performed, showed some errors that became characteristic; therefore the author states the need of speech tests (logo-audiometry) being made specifically, and not only numerically; *i.e.*, each word must be registered by the examiner as repeated by the patient, so as to permit further observation of the results obtained with treatment, as well as to identify certain hearing losses. The following observations were made:

First, Nasal sounds are normally lost in central deafness (An, En, In, On, Un).

Second, The S's are well perceived while they are the first to be lost in the cases of cochlear deafness.

Third, There is great recurrence of mistakes of certain consonants as: T for D; D for T; F for P; P for T; (percentual is given).

Fourth, Frequently fantastic answers are obtained.

c. As a clinical aid to the diagnostic, the Cawthorne-Hallpike test will show directional preponderance in some cortical deafnesses.

d. The author fails to find a differential diagnosis in the cases of deafness by envolvement of structures between the cochlea and the cortex, but assumes that patients with linear tono-audiograms, no recruitment, no directional preponderance and negative encephalographic chart, will in most cases have lesions in the tuberculum quadrigeminus inferior or in

the medical geniculate body or in the nuclei of the acoustic pathway.

DR. HIRSH: Thank you, Dr. Sá. Any other questions or comments?

UNIDENTIFIED DOCTOR: Excuse my very bad English, but I was impressed by what Dr. Sá said, because we made the same investigation, and in Spanish words, to have in the corresponding language different quality deep tones.

DR. GREINER: When you have a very strong amplification your distortion becomes greater than when you have a middle amplification. When you have a peripheral or cochlear distortion this remains the same on every level of intensity. When you have a cortical lesion your distortion first diminishes and then, when the distortion goes up, you have a greater discrimination.

DR. HIRSH: Tomorrow you will hear the physiologists tell us about the cortex, and how they will interpret some of the clinical results of today.

PENDULUMS, TRAVELING WAVES, AND THE COCHLEA:

Introduction and Script for a Motion Picture.*

G. v. BÉKÉSY,

Harvard University,

Cambridge, Massachusetts.

I. INTRODUCTION.

The purpose of this film is to make us familiar with the properties of traveling waves which seem to play a role in hearing. Only transverse waves are described, since the movements of the basilar membrane seem to be of this sort. The vibration amplitudes on the human basilar membrane,

*This film, originally scheduled as a portion of the session on the Physiology of Hearing, was shown Tuesday evening, May 14, in the clinics and laboratories of the Central Institute for the Deaf, St. Louis, Missouri.

during stimulation with even a very strong sound, are so small that it would be very difficult and expensive to photograph them. For this reason the traveling waves are demonstrated on models. These models have the great advantage that they permit us to investigate several different phenomena as a function of a number of variables.

In the beginning of the film it is demonstrated that, even with an oscillating system made up of only two coupled pendulums, the energy of one pendulum does not stay attached to the pendulum in motion, but through coupling, is transmitted to the second pendulum. Naturally, if there are several pendulums coupled together, the energy flows through one pendulum to its neighbor, and in this way a traveling wave is formed. In general, the same holds for membranes or for any system of this type.

Propagation, reflection and standing waves are illustrated, and it is demonstrated that even with a set of coupled resonators, such as those described in the resonance theory of Helmholtz, transients always produce traveling waves.

The film closes with an enlarged model of the cochlea. On the model the nerve supply is represented by the sensory nerves of the human skin. It is the skin which picks up the traveling waves along a plastic membrane. The vibrations felt are similar to those on the basilar membrane of the human ear, and they produce a sensation which is very sharply localized along the membrane even though the maximum of the vibration amplitude distribution is quite flat. Changing the stimulating frequency makes the sensation move up and down on the skin of the arm, thus permitting frequency analysis by place determination.

II. SCRIPT.

This film illustrates the principles basic to the complicated vibration pattern of the inner ear.

Part I shows the production, propagation, and reflection of waves in a system of coupled pendulums.

Part II shows traveling waves similar to those observed in the inner ear.

Part III shows an enlarged dimensional model of the vibrating parts of the cochlea.

Part I—System of Coupled Pendulums.

The pendulum system consists of a series of green balls *B*, each suspended from the supporting rod *A* by a thread (see Fig. 1). Adjacent pendulums are coupled by small orange balls attached between them. When the large driving pendulum is coupled to the supporting rod, it sets the whole series in motion simultaneously; when it is coupled to the series only (by an orange ball) it sets the first pendulum in motion.

In a system of two coupled pendulums, when one is set in motion it transfers its energy to the other, which in turn returns the energy to the first.

As the coupling is increased, by lowering the coupler, the amplitudes of the coupler become larger, and the energy transmission is, therefore, increased.

When there are four pendulums, the energy travels a greater distance from the point of origin.

In a long series of pendulums the coupling transports energy away from the source and forms a traveling wave.

The formation of traveling waves under special conditions.

In the inner ear the driving forces act on large sections of the vibrating system. This distribution of forces can be simulated in the pendulum system by setting the driving rod in oscillation. When all the pendulums are of the same length and all are driven simultaneously, they swing in phase and no waves are produced. The height of the coupling balls is immaterial.

If the driving forces act on only half of the pendulum series, traveling waves develop in the center and spread in both directions. When the distribution of forces is constant, a discontinuity in the lengths of the pendulums will also cause waves to spread in both directions from the discontinuity; however, when the driving frequency is lower than the resonant frequency of the pendulums, no waves are

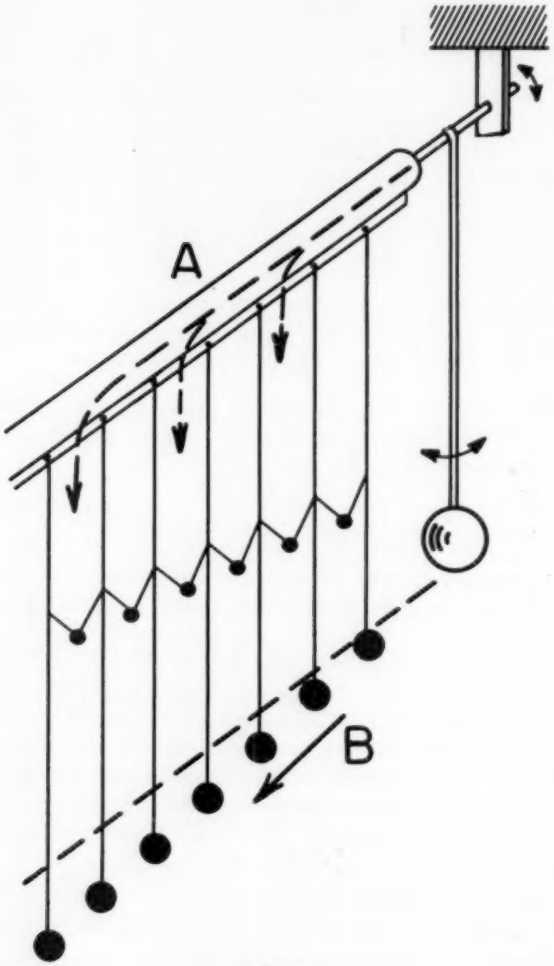


Fig. 1.

formed, even though the force acts on only one end of the series.

Propagation of Waves.

When an oscillation is suddenly transmitted to one end of the coupled pendulum series, traveling waves of varying speed are produced. The high frequency components of the transients produce short waves, and these waves travel faster than the long waves. This is called dispersion. In a dispersive system, two types of velocity can be seen, phase velocity and group velocity.

Phase Velocity—the maximum of the small sinusoidal waves moves quickly from right to left and disappears at the front edge of the group (see Fig. 2).

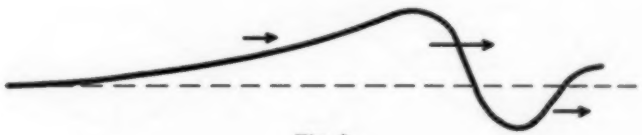


Fig. 2.

Group Velocity—the whole group of sinusoidal waves moves into the region of the undisturbed pendulums at a much lower velocity. With smaller coupling the difference between group and phase velocity is even more pronounced.

When the oscillation is continuous, however, only the phase velocity can be observed. In order for us to see group velocity, transients are necessary. The wave length for a given frequency depends upon the amount of coupling. When the coupling is large, the waves are long. When the coupling is small (the orange balls high) the wave length is small.

Reflection.

Whenever a traveling wave reaches a discontinuity, total or partial reflection occurs and standing waves may appear. Standing waves are characterized by nodes and loops in

fixed positions. Usually the movements of adjacent loops are out of phase (see Fig. 3).

In the picture an electro-magnet holds one of the central pendulums in a fixed position, and standing waves occur between the stationary pendulum and the driving source. The moment the pendulum is released traveling waves occur and move along the whole system. A local discontinuity in a traveling wave produces standing waves as a consequence of reflection.

Part II—Two systems illustrating traveling waves similar to those observed in the inner ear.

A system of pendulums can simulate the vibration pattern observed in the cochlea of a living guinea pig or in a prepara-

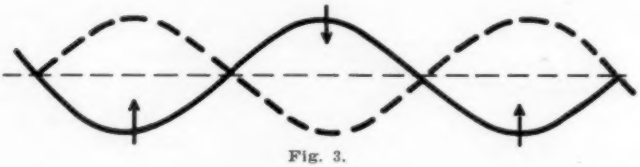


Fig. 3.

tion of human temporal bone. When the lengths of the pendulums decrease continuously from one end to the other, what we observe is traveling waves moving in the direction of the longer pendulums. For the past 100 years Helmholtz's theory has dominated the field of hearing.

Helmholtz Theory.

Lord Kelvin photographed Helmholtz in his laboratory in 1894—the age of kerosene, gaslight, and basic research. According to the resonance theory, there are free resonators in the inner ear, each of which resonates at a different frequency. Without coupling, the pendulum series becomes a system of free resonators. The short pendulums at the far end are fast moving, the long ones at the front are slow moving.

Traditionally, it has been assumed that traveling waves

do not occur in a system of free resonators, but when the whole support of the pendulums is set in oscillation at a high frequency, a traveling wave develops at the onset of the oscillation. Its maximum amplitude is in the region of the shorter pendulums. As the driving frequency is lowered the maximum moves toward the longer pendulums.

Transients are important in the recognition of speech and music. For transients there is almost no difference between the resonance and traveling wave theories of hearing.

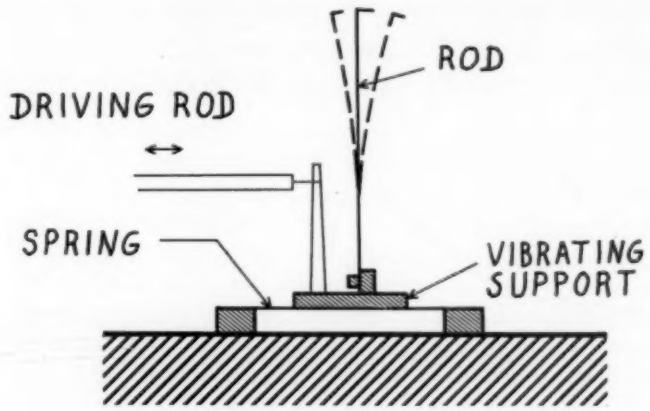


Fig. 4.

Reed System.

In the following pictures, the same phenomena are illustrated with a series of reeds attached to a supporting base which can be set in vibration by a driving rod. The reeds are tuned at equal intervals and their resonant frequencies are within the range of hearing. The reeds in front are stiff and resonate at higher frequencies, those in back at lower frequencies (see Fig. 4).

Applying a sinusoidal vibration to the support sets the reeds in motion. Reeds that resonate at frequencies close to

the driving frequency vibrate at the largest amplitudes. As the driving frequency is lowered, the region of maximum vibration shifts toward the low-frequency reeds.

The reeds shown vibrate 60 times per second, and only a blur could be seen, but when the pictures are projected 100 times slower, the movements of each reed can be followed. The white line made by the tips of the reeds describes a vibration pattern characteristic of free resonators.

The reeds below the resonance point—the point of maximum vibration—are vibrating 180 degrees out of phase with the reeds above the resonance point (see Fig. 5).

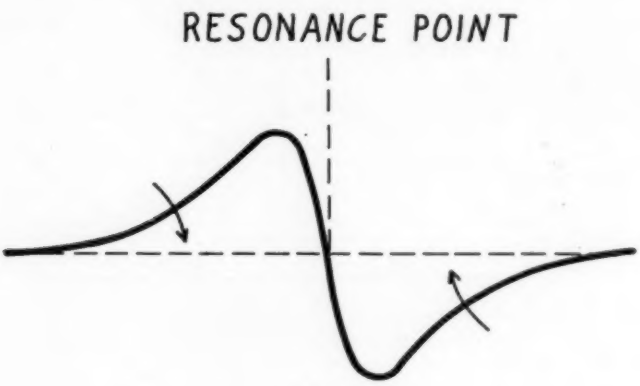


Fig. 5.

In spite of the assumption that traveling waves do not occur in a system of free resonators, they can be seen forming during the onset and termination of a periodic driving force. The resonance theory of hearing assumes the vibration pattern you have just seen; that is, resonating elements vibrating freely.

In the basilar membrane, however, each element is coupled to its neighbor. This coupling is simulated here by inserting a rubber band along the reeds. With the reeds thus coupled, displacement of one reed moves its neighbor.

Because the driving rod is atached to the supporting base, all the reeds are exposed to the same driving force. As in the coupled pendulum series, traveling waves are present, even for continuous vibrations. In order to avoid reflections, damping has been added in the form of a piece of sponge rubber. At normal speed we would see only the amplitudes of the vibrations moving along the row of reeds.

The traveling waves are similar to the waves produced in the cochlea of an anesthetized guinea pig or in a preparation of the human cochlea, but in the cochlea there is better damping.

In an elastically coupled reed system, a traveling wave does not lose energy as it travels. When the wave strikes an obstacle, such as the end of the reed system, it is reflected, and standing waves result. The same phenomenon is shown at normal speed and in slow motion. The adjoining parts of the basilar membrane are coupled in part by the fluid surrounding the membrane.

If we remove the rubber band from the reed system and immerse the reeds partially in water, the resulting frictional coupling produces traveling waves similar to those produced with elastic coupling. The next picture shows the combined effects of both types of coupling. As the frequency is lowered continuously, the point of maximum amplitude moves in the direction of the low-frequency reeds.

Paradoxical Waves.

The lower part of the next picture shows the wiring diagram of a low-pass filter; the upper part represents the voltage across each section. The waves move from left to right, but with a high-pass filter the waves move from right to left, that is, toward the source of the energy. Waves traveling *toward* the source are common in electrical networks but are rarely seen in nature. Experiments show that they can occur in the cochlea.

We can duplicate these paradoxical waves in the reed system by exerting a driving force on a whole section of damped reeds simultaneously. When this is done, the driving

force can be moved a certain distance along the reeds without changing the *direction* of the waves.

Part III—A model of the cochlea with nerve supply.

It is hard to imagine how traveling waves act on the sensory nervous system. Our best approach is through an experiment. With dimensional analysis we can make models of ships and airplanes that simulate all the phenomena of full-scale vessels. In the same way we can contruct an *enlarged model* of the cochlear duct that incorporates the mechanical data of the human cochlea. Several such models have been constructed, of which the most recent is in Göttingen.

The model used here consists of a plastic tube with a membrane that is thicker at one end than at the other. The tube is filled with water and at the thick end contains a vibrating piston corresponding to the stapes footplate.

Sinusoidal vibrations produce traveling waves on the membrane. The maximum of the vibration is close to the piston (stapes) for higher frequencies.

When the forearm is placed on the vibrating membrane, even though the maximum is broad, the sensation is concentrated in a small region of the skin. As the frequency changes the locus of the sensation moves along the arm.

The subject moves a pointer along his arm to show the locus of the sensation for a given frequency. The sensation extends over only 2 or 3 cm., as is shown by the small oscillating movements.

Preparations of the cochlea show that there is in the inner ear a vibration pattern with a broad maximum whose locus changes with frequency, and experiments indicate that the nervous system sharpens this maximum.

At the low frequencies the nervous system can perform a frequency analysis by responding directly to the frequency of the vibration, but at the middle and higher frequencies only the locus of the maximum determines the pitch.

The impressive factor of this neuro-mechanical model is

that it can perform a frequency analysis much more quickly than can any known electrical filter system.

The locus of the sensation on the skin is determined with equal accuracy whether the frequency is presented for a second, or for as little as 2 cycles—which in this experiment is 0.02 second.

For all our technical skill when nerves are involved, nature is far ahead of us.

Acknowledgment.

The electronic equipment used in these experiments and the shooting of the film were achieved with the collaboration of Rufus L. Grason.

REFERENCES.

Background information concerning the patterns of traveling waves in the cochlea and on the conditions of appearance of traveling waves will be found in the following articles:

BÉKÉSY, G. v.: Description of Some Mechanical Properties of the Organ of Corti. *Jour. Acoust. Soc. Amer.*, 25:770-785, 1953.

BÉKÉSY, G. v.: Paradoxical Direction of Wave Travel along the Cochlear Partition. *Jour. Acoust. Soc. Amer.*, 27:137-145, 1955.

BÉKÉSY, G. v.: Human Skin Perception of Traveling Waves Similar to Those on the Cochlea. *Jour. Acoust. Soc. Amer.*, 27:830-841, 1955.

BÉKÉSY, G. v.: Simplified Model to Demonstrate the Energy Flow and Formation of Traveling Waves Similar to Those Found in the Cochlea. *Proc. Nat. Acad. Sci.*, 42:930-944, 1956.

BÉKÉSY, G. v.: Current Status of Theories of Hearing. *Science*, 123:779-783, 1956.

BÉKÉSY, G. v.: Sensations on the Skin Similar to Directional Hearing, Beats, and Harmonics of the Ear. *Jour. Acoust. Soc. Amer.*, 29:489-501, 1957.

BÉKÉSY, G. v.: Neural Volleys and the Similarity Between Some Sensations Produced by Tones and by Skin Vibrations. *Jour. Acoust. Soc. Amer.*, 29:1059-1069, 1957.

BÉKÉSY, G. v.: Funneling in the Nervous System and Its Role in Loudness and Sensitivity on the Skin. *Jour. Acoust. Soc. Amer.*, 30:May Issue, 1958 (In Press).

WEDNESDAY MORNING SESSION,

May 15, 1957.

PHYSIOLOGY OF HEARING

The International Conference on Audiology reconvened at 9:30 A.M., in the Auditorium of the Washington University School of Medicine, St. Louis, Missouri; Dr. Harvey Fletcher, Director of Research, Brigham Young University, Provo, Utah, presiding as Chairman.

DR. FLETCHER: We are to discuss today how the ear works, and the physiology of hearing. The transmission of sound to the ear, through the middle ear, and into the cochlea, has been divided into two parts: first, that part concerned with the process of sound conduction through the middle ear; second, transmission in the cochlea.

PROCESS OF SOUND CONDUCTION.*

MERLE LAWRENCE,

University of Michigan,

Ann Arbor, Mich.

Many of us involved in the field of hearing do research out of curiosity. We would like to know how the ear works. The clinical otologist, on the other hand, is interested in how the ear works because he would like to know what to do when infection or trauma interfere with normal function. Specifically, he would like to know what certain steps that he might take would improve the performance of this disarranged mechanism; so the more basic knowledge revealed by the

*From the Physiological Acoustics Laboratory, University of Michigan, Ann Arbor, Mich. Preparation of this report was supported by the Research and Development Division, Office of The Surgeon General, Department of the Army, under contract No. DA-49-007-MD-634, and by the funds for Research in Human Resources, University of Michigan.

curious, the better off is the clinician, and there is no other area of auditory function that has so clearly shown this picture of basic knowledge contributing to successful clinical procedures than studies of the sound conduction process of the middle ear.

We see the successful beginning of middle-ear surgery back in 1890[6] with attempts at mobilizing ankylosed stapedes. Through many variations this procedure was replaced by the fenestration operation, first tried in one form or another around 1900,[7] and finally perfected to a single stage, and highly successful method, by Lempert some 15 years later.[8] Actually it was the success of this operation that inspired some concentrated research on the conductive processes of the ear, and as a result of the subsequent understanding of the middle-ear mechanics there has been developed a number of operative procedures for the reconstruction of chronic middle-ear conditions that up to now caused deafness that had to be lived with.

Our purpose here is to consider the way sound is transmitted through the middle ear and the principles of sound conduction that are involved. Because of time limitations the discussion is confined to two topics, the mechanical operation of the middle ear, and the fidelity with which the middle ear performs.

Of the peripheral structures the sensory cells of the organ of Corti are the most important. To have hearing these cells must be vibrated, and to survive they must be nourished. Because of their sensitivity to vibrations they cannot be nourished by any system that contains elements that might pulse or produce rapid movements through turbulent fluid flow, so we find these cells surviving without any direct blood supply but by being immersed in a fluid medium which moves imperceptibly, bringing nutrient material to the cells and carrying away waste products. Hearing results when the desired vibrations are introduced into this fluid medium and are effectively transmitted to the cells.

By placing a needle electrode in the vicinity of the cochlea, and recording the potentials that arise from vibrations of the

sensory cells, it is possible to obtain a measure of the efficiency of vibrations introduced into the inner-ear fluids. Since there is no threshold or level of sound at which the potentials suddenly appear, it is necessary to use some constant voltage output in response to sounds of different frequencies as a measure of the ear's sensitivity. By recording the sound pressure necessary to produce this pre-established level of response at the various frequencies in the normal ear, and comparing this with the responses under experimentally altered conditions, it is possible to obtain a quantitative measure of the effects of the abnormal conditions.

For the purposes of simplifying the analysis of middle-ear function let us start with the condition in which there is no middle ear, the inner ear being bounded on the lateral side by the oval and round windows. Our first concern is whether one of these air-conduction routes is more efficient than the other.

Sound is first conducted by means of a small probe tube to the oval window and the sound pressure necessary to produce our pre-established response level measured by means of a calibrated probe-microphone system. The sound is then conducted to the round window and these measurements repeated. Fig. 1 shows the result of such an experiment. As is to be expected, considerably more sound pressure is needed to produce the required response when the middle ear is removed than when it is present, but it makes very little difference for effective vibration of the inner ear whether the sound is introduced into the oval window or the round window.

Because the two windows lie at opposite ends of a fluid system, it has long been suggested that a back and forth movement of this fluid column is essential for sensory-cell stimulation. This can be determined by carrying the above experiment a step farther. While the sound is being introduced into the oval window the round window can be blocked. This is a rather difficult thing to do effectively because the actual volume displacement of the fluids of the inner ear is so minute. It amounts to the volume of a sphere with the diameter of a red blood cell when the ear is stimulated by a sound of 1000 c.p.s. at 74 db above .0002 dynes/cm^2. An air

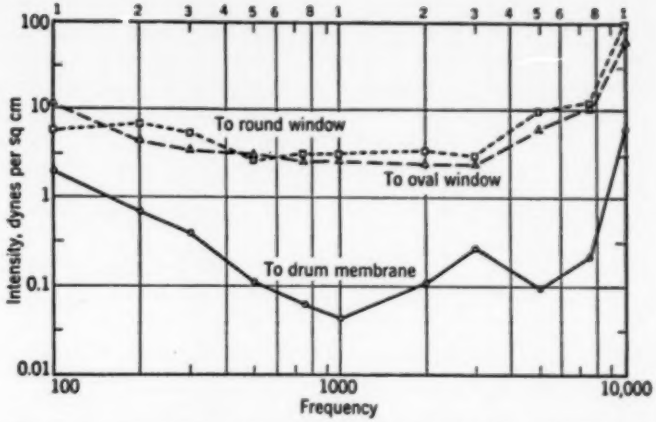


Fig. 1. A comparison of oval-window and round-window pathways after the middle ear has been removed. The ordinate shows the sound pressure necessary to produce 10 microvolts of response at the indicated frequencies; 1 dyne/cm² is equivalent to 74 db. above .0002 dyne/cm². From Reference 10.

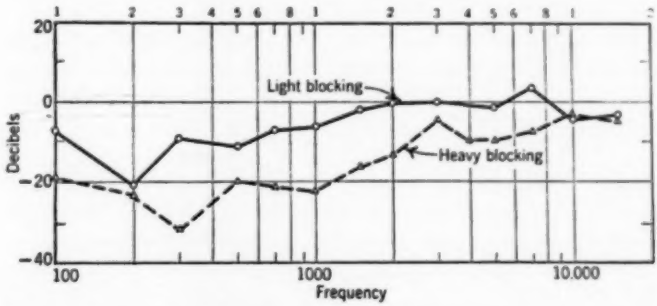


Fig. 2. The effect of blocking the round window upon the response to sound delivered via the oval window. From Reference 10.

bubble one million times this size, about one-third of a cubic millimeter, would allow almost complete freedom of the fluid movements.

Fig. 2 presents the result of such an experiment. The effects of two degrees of blocking are graphed to show something of the range of effects that can be obtained. The decre-

ment is greatest for the low frequencies, and is in excess of 20 db for those below 1000 c.p.s. It is necessary to dry the surfaces and pack the niche completely with wax in order to produce the results shown. It is apparent that the oval window pathway is effective only as long as the round window is free at the other end of the system.

We find also that the oval window must be free to make the round window pathway effective. Because of the presence of the footplate of the stapes it is easier to block the oval window than the round window, and by embedding the stapes in wax the results of Fig. 3 are produced. Now the effects

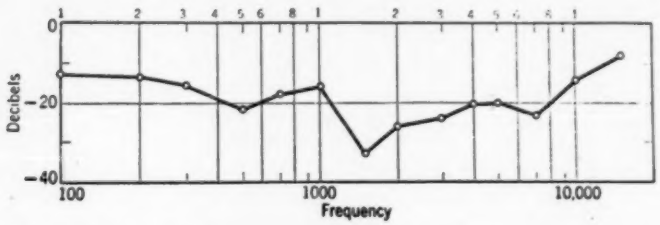


Fig. 3. The effect of blocking the oval window upon the response to sound delivered via the round window. From Reference 10.

are most severe in the range immediately above 1000 c.p.s., indicating that the method of blocking the two windows has changed the frequency response properties in different ways, but the principle is still demonstrated that in order for vibrations entering by one window to be fully effective in stimulating the sensory elements, the other window must be free to act as a relief opening at the other end of the system. This was a theory first expressed by Edward Weber[8] more than a century ago.

This principle can be studied more quantitatively if we introduce sound into both windows at the same time. Such a situation would exist in an individual with no tympanic membrane or ossicles, so that both windows were exposed to the same sound field. Visualize the alternating positive and negative sound pressure of a sound wave reaching one of the windows as pushing the membrane in during the positive

pressure phase and pulling it out during the negative pressure phase. If both windows are being pushed in at the same time the situation would be the same as blocking one of the windows, because no relief opening is available for either source of sound. This condition is portrayed schematically in Fig. 4. It can be seen that in such a phase relationship no movement of the basilar membrane with subsequent sensory-cell stimulation is possible. This is equally true of the phase

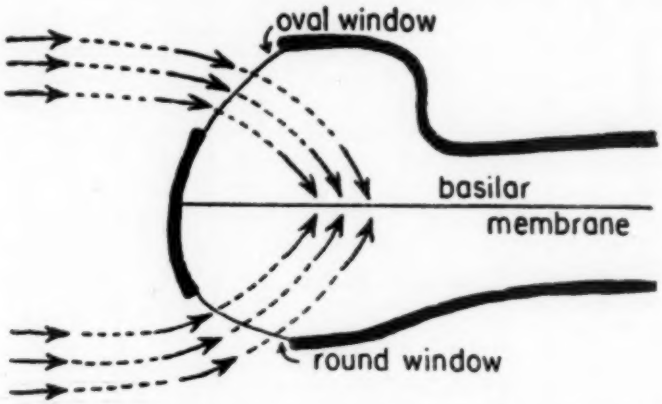


Fig. 4. Schematic representation of sound entering both oval and round windows at the same time. The phase condition represented here is such that there would be complete cancellation of response. The sounds are in phase at the two windows and of equal intensity. From Reference 11.

relation when both windows are being pulled out at the same time; however, when one window is pushed in by a positive sound pressure and the other window is pulled out by a negative pressure brought about by delaying the sound by one-half a wave length, the effectiveness of the relief opening is enhanced. By such a situation the amplitude of vibration of the basilar membrane should be double that produced by sound in one window alone.

Again, by recording the electrical responses of the cochlea it is possible to obtain a quantitative measure of the above. Sound from one electronic oscillator is used with the output

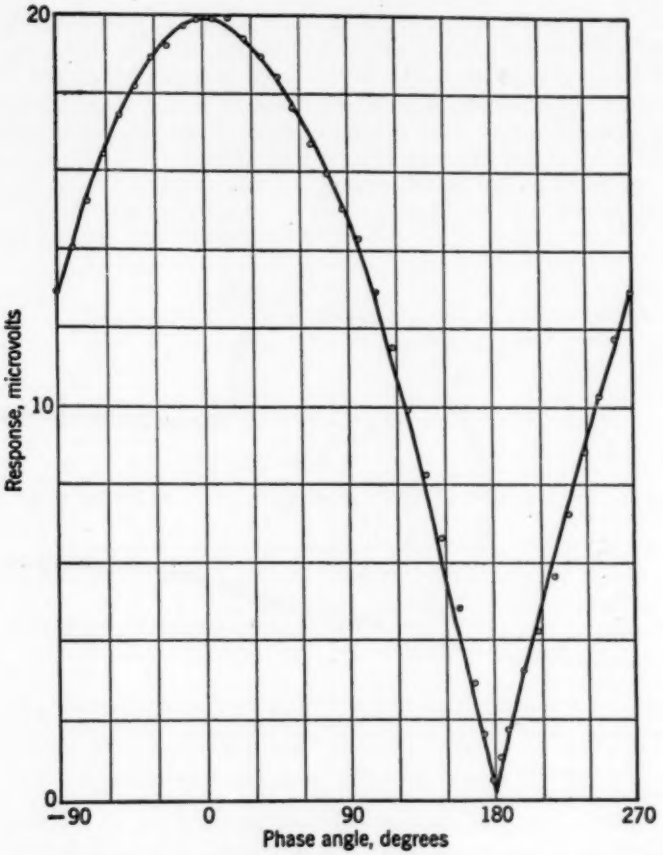


Fig. 5. Vectorial summation in the cochlear response to a tone introduced simultaneously by way of the oval and round windows. The phase relation of the two pathways was varied over a range of 360°, as shown on the abciasa. The points represent observations and the curve represents theoretical summation. From Reference 10.

passed through a dividing network. Into one channel a phase changer is introduced and the two sources separately led, by means of small tubes, to each window. The sound level is again recorded by a calibrated probe-microphone system, and the recorded electrical potentials serve as a measure of the effectiveness of the stimulus. Fig. 5 shows the magnitude of

the cochlear response as the phase of the sound in one window is shifted with respect to the phase of the sound in the opposite window. The sound is first directed into one window alone and set at a sound pressure sufficient to give rise to a response of 10 microvolts. This tone is then shut off and that directed toward the opposite window set to give a 10 microvolt response. For the readings shown in the figure the tones are introduced into the two windows simultaneously. The response shows what is to be expected from the vectorial sum of two sine waves of equal intensity when one is varied in phase. As expected, in a phase relationship where one window is pushed in while the other is pulled out the response is double what sound in one window alone would give, and when they are 180 degrees from this condition, that is, both windows being pushed in or pulled out simultaneously, the response is completely cancelled. The interesting thing to note is that as the phase varies through the complete 360 degrees, the two-window response is as good or better than that from one window alone over a phase difference of 240 degrees, while the two-window response is worse over only 120 degrees. It is not likely that the sound, under conditions where both windows are exposed, hits them in the exact phase relation for cancellation. In the first place the windows are not in exactly the same plane, considered from the point of view of the sound-wave front, and secondly, one window contains a stapes or footplate and the other a membrane. These introduce phase differences, as will be shown shortly, and it does not take much of a phase shift to bring the response up to a level of that given by stimulation through one window alone. If the two windows were exactly alike they would have to be separated by a distance of about seven inches for a frequency of 1000 c.p.s. In the scheme behind our phylogenetic development this apparently was considered impractical.

Another factor influencing this two-window summation is the intensity of the tones introduced. Complete cancellation depends upon equal intensities, so this unwanted condition can also be avoided by the introduction of a more intense sound in one window than the other. To accomplish this, advantage has been taken of a fact which make a fluid-filled

ear quite impractical in an air environment, as was the case some 250 million years ago, when the predecessors to the amphibians first found themselves on land.

Because of the extreme differences in the properties of elasticity and density between air and liquid, approximately 99.9 per cent of the acoustic energy is reflected back at an air-water interface. This means that there is a loss of 30 db in transmitting the vibratory energy from the air to the inner-ear fluids. By the use in one window only of a mechanism that matches these properties of air and water, it is possible to overcome both of the disadvantages of the two-window

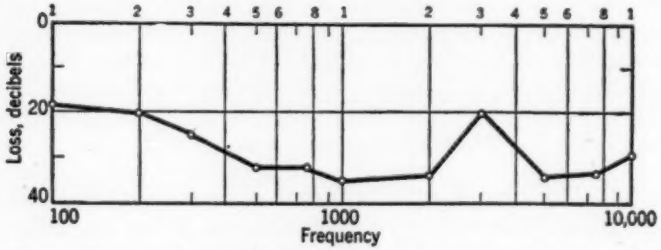


Fig. 6. The loss of sensitivity resulting from the removal of the middle ear mechanism in the cat. From Reference 10.

fluid system: *1.* the cancelling effect of equal-intensity, in-phase sound entering by the two windows is overcome by increasing the intensity in one window by 30 db over that in the other; *2.* the loss in transmitted energy due to the air-liquid interface is overcome by matching the properties of air to those of the denser liquid, and this apparently is just what the middle ear does.

To see whether the middle ear is actually performing this function, and if so, how efficiently it is accomplishing the impedance match, we can again resort to an animal experiment, recording the electrical responses of the cochlea under conditions in which the ear is normal and those in which the middle ear is removed. Fig. 6 represents the loss, in the cat ear, resulting from removal of the middle ear structures up to the stapes. The loss is not the same for all frequencies,

because of the resonant properties of the middle-ear structures, but the average is approximately 28 db, which is close to the theoretical loss of 30 db. It means that, considering the errors which can be attributed to the nature of the experiment, the middle ear of the cat is almost perfectly efficient.

Since the time of Helmholtz much research and thought have gone into the problem of how the middle-ear structures accomplish this match. Of the theories of action that have been proposed, two have been shown experimentally to contribute to the impedance matching process.

The first of these depends upon the *areal ratio* between the tympanic membrane and the footplate of the stapes. In man the average of the available data shows this ratio to be about 21 to 1. Considering the fact that the tympanic membrane is not like a piston, but rather like a stretched membrane attached around the edge, the effective area of the membrane must be reduced by about one-third, making the ratio 14 to 1.

The second characteristic of the middle ear that aids in the matching process is the *lever ratio* brought about by the peculiar arrangement of the ossicular chain. Measurements of this in the human are few and probably not very accurate. Dahmann[3] made what are probably the most reliable measures, by placing small mirrors on the drum membranes and then on the long process of the incus. The ratio so determined was found to be small: about 1.3 to 1.

The product of the areal ratio and the lever ratio give us the transformer ratio of the entire middle ear, which then amounts to approximately 18 to 1. This pressure increase at the footplate of the stapes is equivalent to 25 db, which is not enough to overcome completely the theoretical loss of 30 db., but these figures should not be taken too seriously because of the limitations in both the experimental data and theoretical calculations. In calculating the theoretical 30 db loss in the passage of sound from air to the fluids of the inner ear, data for boundless media were used, whereas the air of the middle-ear cavity or fluids of the inner-ear space are far from boundless. On the other hand, data used to determine the areal ratio are limited, and the data of the lever ratio may

not be very accurate because of the difficulty of determining accurately from an optical lever the excursion of a vibration when the axis of rotation of the mirrors is not known; nevertheless, the figures are close enough to indicate that the principles described are the right ones.

Measurements of the transformer ratio, which have been made on human cadaver material, show that these figures are fairly accurate. Békésy[1] drained out the fluid of the inner ear, dissected away the cochlea, and then applied sound to the inner surface of the stapedial footplate. This he adjusted in intensity and phase so as to cancel the effects of another

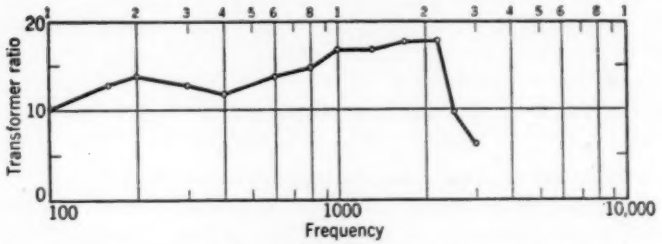


Fig. 7. The transformer ratio of the human (cadaver) ear according to Bekesy[1]. From Reference 10.

sound of the same frequency introduced at the drum membrane. A capacitative probe near the surface of the stapes showed when cancellation was complete. From these he obtained a transformer ratio of 10 at 100 c.p.s., rising to 18 at 2200 c.p.s. and then rapidly falling. This is shown in Fig. 7. Now the lever ratio and areal ratio of the middle ear will not change with frequency, so the variations in the transformer ratio can probably be attributed to the influence of the inherent response properties of the ossicular chain.

At this point we must mention some other interpretations made on the basis of another experiment reported by Békésy in 1942.[2] In this instance he measured, also on cadaver specimens, the volume displacement of the round window for a number of conditions. In the first of these he cancelled out a tone applied to the inner ear through the intact middle-ear

structures by a tone of the same frequency, but adjustable in intensity, applied through the round window. A determination of the current in the cancelling receiver gave a measure of the volume displacement. He then repeated the balancing process after removing all of the middle-ear structures, including the stapes and round window, by conducting the sound directly to the fluids of the inner ear through the oval and round window openings. These results showed considerably larger volume displacements at low frequencies than before, but their magnitude showed a progressive decrease as the frequency was raised until, at the higher frequencies, they became smaller than in the intact ear.

Fletcher[4] divided the first set of values by the corresponding ones in the second set, and regarded the quotient as representing the transformer ratio of the middle ear. The calculations, which he interpreted as demonstrating a middle-ear pressure reduction for frequencies below 650 c.p.s., are presented as curve *a* of Fig. 8. He saw advantages in this sort of action in that the system would discriminate against disturbing low frequencies, and on the basis of this it has been suggested that the middle ear does not at all act like an impedance-matching transformer; however, in this same series of experiments, Békésy made some other measurements that more accurately reflect the action of the middle ear. In this instance the second measurements were made with the drum membrane and outer ossicles removed but with the stapes left in the oval window and with the round window membrane intact.

When the values of volume displacement for the intact ear are divided by those for the above conditions we have a curve, *b* in Fig. 8, that is in better accord with the earlier results and testifies to a useful transformer action of the middle ear for all frequencies.

It is obvious from these determinations, and those showing the contribution of the middle ear to sensitivity, that the advantages are not the same for all frequencies. The middle ear is not a truly high-fidelity device, but it does remarkably well. Three forms of distortion, found in all forced-to-

vibrate devices, that have been extensively studied in the middle-ear response to acoustic vibrations are frequency, phase and non-linear or amplitude distortion.

The first of these, frequency distortion, is illustrated in Fig. 6. This curve represents the frequency response for the middle ear of the cat. Like any other device, when forced to vibrate, it exerts its own resonance properties into the vibration and so alters the frequency characteristics. In the human ear this middle-ear frequency distortion is re-

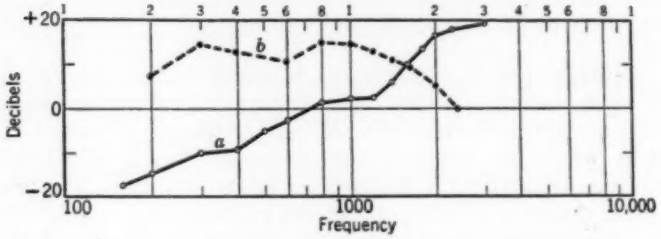


Fig. 8. The pressure amplification afforded by the middle-ear mechanism as derived from the observations of Bekesy.[2] Curve *a* shows the results of Fletcher's computations and curve *b* those of Wever and Lawrence[10]

flected, along with many other hearing phenomena, in the sensitivity curve.

Whenever there is frequency distortion due to the characteristic mass and stiffness properties of the driven structure, phase differences are also introduced into the vibratory response. This means that as the sound at the tympanic membrane reaches a positive-pressure peak or a negative-pressure peak the ossicular chain lags behind or, in fact, may appear to precede the movements of the air-borne vibration. Thus the chain of middle-ear bones introduces phase distortion, not only by shifting the phase but also by doing it differently and in varying amounts over the frequency range.

By employing a stimulating system similar to that described for studying the effects of different phase relations at the oval and round windows we can find out how much the middle ear shifts the phase for various stimulating tones.

Fig. 9 shows the phase shift introduced for frequencies from 100 to 10000 c.p.s. by the entire middle ear. Positive values indicate a phase lag introduced by the ossicular chain. The amount of phase variation is quite moderate for frequencies below 5000 c.p.s., and then grows greater and more variable for the higher frequencies. Actually these phase changes remain constant at the different frequencies and so, of course, make no noticeable difference to hearing. Since the sound

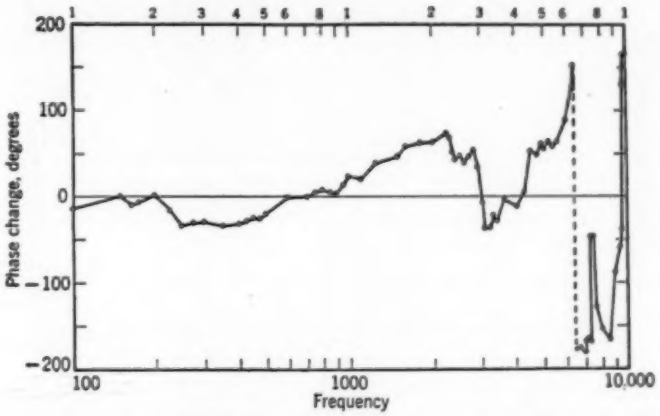


Fig. 9. Phase changes produced by the entire middle-ear apparatus. Positive degrees represent a retardation of phase. From Reference 10.

being transmitted by this route to the oval window is more intense by about 30 db than that reaching the round window, any inner-ear effects due to the phase difference are negligible. When the middle ear is removed, however, so that this advantage is lost, any phase shift in one window relative to the other makes a great deal of difference, as was described earlier.

Measurements made of phase changes attributed to action of the stapes are shown in Fig. 10. These variations are small, never exceeding 15 degrees below 5000 c.p.s., but as shown in Fig. 5 it takes very little shift from the cancellation point to improve the situation. This demonstration of the

phase characteristics of the stapes serves to emphasize the importance of establishing a favorable phase relationship between the two windows during any reconstructive surgery of the middle ear performed for the purpose of improving the hearing.

The third type of distortion concerns the ability of the structures to follow increases in amplitude of the driving

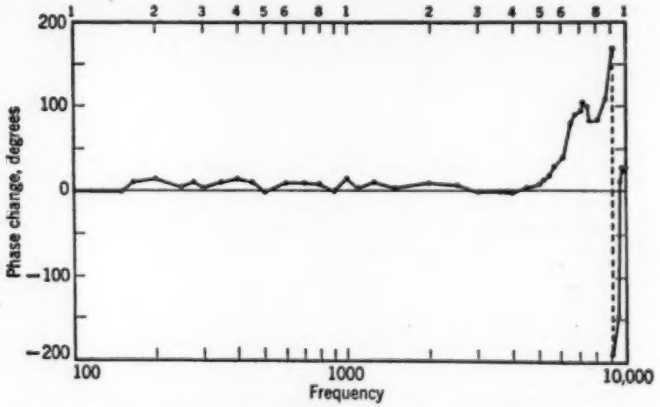


Fig. 10. Phase changes produced by the stapes. Positive degrees represent a phase lag in the oval-window pathway relative to the round-window pathway. From Reference 10.

vibration. A characteristic of the electrical response of the cochlea in response to a pure tone is that as the sound amplitude is increased the electrical output increases proportionally over a characteristic range, and then departs from this straight-line function until finally it reaches a maximum. The point at which the response becomes no longer proportional to the amplitude increases of the stimulating sound is called the "limit of linearity," and the point at which there are no increases in response to further increases of stimulus is called the "maximum," as shown in Fig. 11. The electrical response recorded in this instance reflects not only the transmission of sound through the middle ear and through the fluids of the inner ear, but also the process by which this

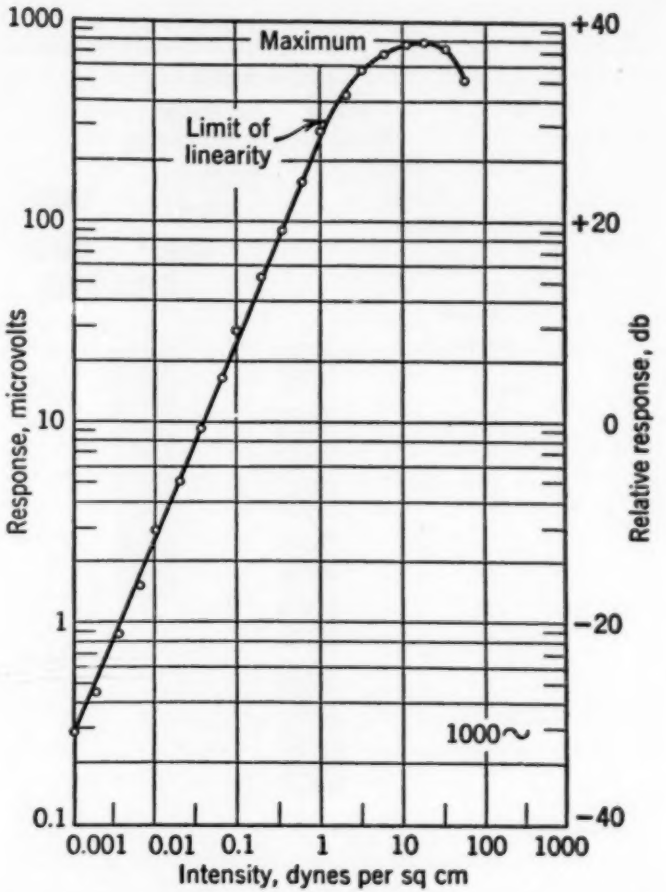


Fig. 11. The intensity function for the cochlear response recorded from the round window of the cat. The "limit of linearity" denotes the point in the response where it is no longer proportional to changes in the stimulus intensity. From Reference 10.

vibration is transformed into the recorded electrical response. We would like to know then, what structure or process brings on this departure from linearity and specifically, for our discussion here, we would like to know whether the conduction process in the middle ear is responsible for this.

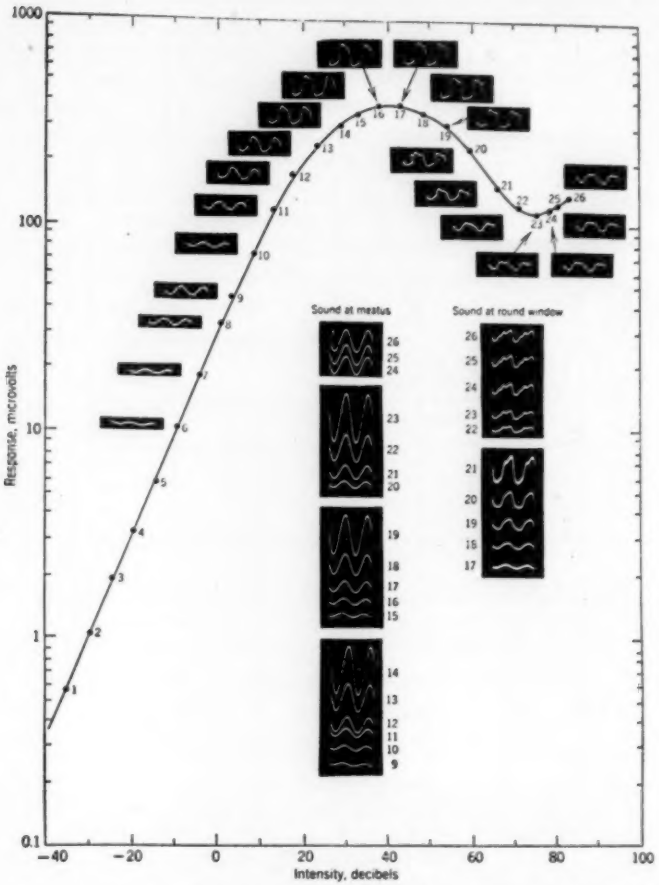


Fig. 12. The intensity function for the cochlear responses of the cat's ear, with a display of wave forms of this response at different levels. The intensity scale is in decibels relative to 1 dyne/cm² (74 db above .0002 dyne/cm²). The points of measurement along the curve are numbered, and from 6 on, each has adjacent to it an oscillogram of the cochlear response obtained. For these waves the amplification of the oscillograph was constant up to point 9, and then was reduced and held constant thereafter for points 10 to 26. In two columns below are oscillograms for some of the sounds entering the ear at the meatus and also for sounds picked up from the round window. In recording these sounds the amplification of the oscillograph was constant for each block of pictures but was varied between blocks. From Reference 10.

The evidence gathered over the last few years has shown conclusively that the inner ear is responisble for this early departure from a linear response. One way to study this is to record the magnitude of the electrical response as well as its wave shape, and compare this to the wave shape of the stimulating sound and that of the sound after it has been relayed by the ossicular chain to the fluids of the inner ear and escapes from the round window. A comparison is made in Fig. 12. The wave shape of the sound coming from the round window is still undistorted to a level of stimulating sound that has brought about severe wave form distortion in the electrical response. If distortion had occurred in the middle ear this would have been transmitted through the fluids to the round window.

Another test for the locus of this amplitude distortion has been made by applying a vibrator, first to the umbo of the malleus then to the head of the stapes, and recording the cochlear response in both instances. If the middle ear is responsible for this type of distortion, departure from linearity should occur at a lower response level when the middle ear is present than when it is removed. Results are shown in Fig. 13. The limit of linearity occurs at the same response level for both methods of stimulation, indicating that the middle ear is not responsible. Since the middle-ear lever, which reduces stapes amplitude while increasing pressure is removed in one instance, the response through the stapes alone is greater for equal sound pressures than the response after traversing the ossicular chain.

It is true that if the middle ear is driven hard enough it, too, will become non-linear but not until a level of sound has been reached that would have destroyed the inner ear, and nothing seems to affect this ability of the middle ear to respond linearly over a conisderable range of sound intensities. The middle ear can be filled with fluid or the stapes ankylosed and, although sensitivity is lost, the middle-ear dynamic range is very little affected.

In the process of conducting sound from the external meatus to the fluids of the inner ear the tympanic membrane and ossicular chain do not only an amazingly efficient job of

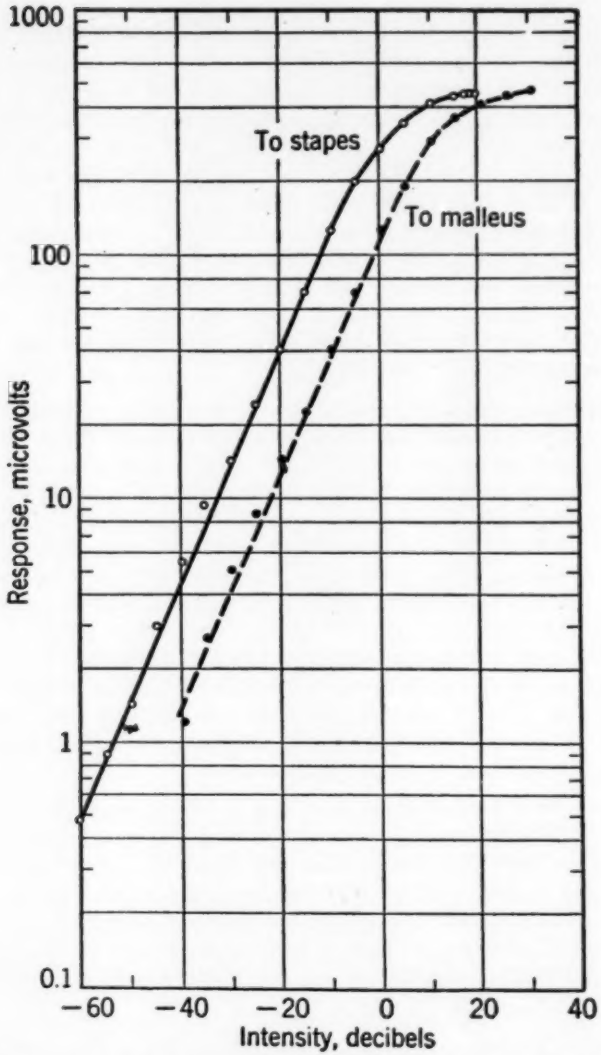


Fig. 13. The cochlear response recorded from the round window in response to mechanical vibration at a frequency of 1000 c.p.s. of the stapes and the malleus. The "limit of linearity" occurs at the same response level in both instances. From Reference 10.

matching the properties of air to those of the dense perilymph so as to introduce sound with a minimum of loss, but do so over a wide range of frequencies with no amplitude distortion within the range of inner-ear response and with tolerable frequency and phase distortion.

REFERENCES.

1. BÉKÉSY, G. VON: Über die Messung der Schwingungsamplitude der Gehörknocheichen mittels einer kapazitiven Sonde. *Akust. Zeits.*, 6:1-16, 1941.

2. BÉKÉSY, G. VON: Über die Schwingungen der Schneckentrennwand beim Präparat und Ohrenmodell. *Akust. Zeits.*, 7:173-186, 1942; trans., The Vibration of the Cochlear Partition in Anatomical Preparation and in Models of the Inner Ear. *Jour. Acoust. Soc. Amer.*, 21:233-245, 1949.

3. DAHMANN, H.: Zur Physiologie des Hörens; Experimentelle Untersuchungen über die Mechanik der Gehörknöchelchenkette, sowie über deren Verhalten auf Ton und Luftdruck. *Zeits. f. Hals-Nasen-Ohrenheilk.*, 24:462-497, 1929; and 27:329-368, 1930.

4. FLETCHER, H. The Dynamics of the Middle Ear and Its Relation to the Acuity of Hearing. *Jour. Acoust. Soc. Amer.*, 24:129-131, 1952.

5. LEMPERT, J.: Improvement of Hearing in Cases of Otosclerosis: a New One-Stage Surgical Technique. *Arch. Otolaryngol.*, 28:42-97, 1938.

6. MIOT, C.: De la Mobilisation de l'Etrier. *Rev. de Laryngol. Otol., Rhinol.*, 10:49-64, 83-99, 113-130, 145-162, 200-216, 1890.

7. SIEBENMANN, F.: Traitement Chirurgical de la Sclérose Otique. XIII Internat. Cong. Med., Sec. d'Otol., Paris, 1900, 170-177; and Ann. des mal. Oreille, 26, ii:467-474, 1900.

8. WEBER, E. F.: Über den Zweck der Fenestra Rotunda und die Vorrichtung der Schnecke im Gehörorgane der Menschen und der Säugethiere. Amtlicher Bericht Versamml. Deutsch. Naturf. Aerzte, 19te Versamml. Gesellsch. Deutsch. Naturf. Aerzte, 1841; Braunschweig, 83-84, 1842.

9. WEVER, E. G., and LAWRENCE, M.: "Physiological Acoustics," Princeton University Press, Princeton, N. J., 1954.

10. WEVER, E. G., and LAWRENCE, M.: The Acoustic Pathways to the Cochlea. *Jour. Acoust. Soc. Amer.*, 22:460-467, 1950.

DISCUSSION OF DR. LAWRENCE'S PAPER ON "PROCESS OF SOUND CONDUCTION."

HENNING E. VON GIERKE.*

The starting point for any serious discussion is a point of disagreement, of doubt, or a question mark. It would be very hard to pick out such a point from Dr. Lawrence's excellent presentation. I think it became quite clear from his

*Aero Medical Laboratory, Wright Air Development Center, Wright-Patterson Air Force Base, Ohio.

presentation that our physical understanding of the conduction mechanism is fairly good, that we are in the position to give average quantitative numbers to the different components of the transmission system, and that in this part of the auditory mechanism the various hypotheses have been almost completely replaced by measured facts. This does not mean that there is not much room left for very worthy and interesting refinement. It simply means that there are no big surprises waiting to be discovered.

Let me add a few words to refine our picture of the physical purpose of the middle ear. Since the acoustical properties of human tissue are very close to the acoustical properties of water, a very simplified statement of the middle ear's task is that it has to match the impedance of air to the impedance of water. (Under impedance we understand the ratio of the driving sound pressure to the velocity of the resulting motion, a measure of mobility. Depending on the phase between pressure and velocity, the impedance can be a mass reactance, an elastic reactance or a frictional resistance, or a combination of these three elements.) This impedance of water or tissue is a well known constant only if we have undisturbed sound propagation in an almost infinitely large medium. This is practically never the case for sound in tissue and, therefore, the impedances of the middle and inner ear do not so much depend upon the acoustical constants of the tissue as they do upon the geometrical shape and the boundaries of the elements involved. For example, a blocking of the round window with an otherwise completely intact conduction mechanism would change the impedance or mobility at the oval window considerably, and consequently also the impedance at the tympanic membrane.

To illustrate this point, impedance values measured at various locations in the ear and on the body surface are shown in Fig. 1, together with the impedance of an infinite water or air medium. Curve a, represents the impedance of a small circular area (of approximately the same size as the tympanic membrane) at the surface of soft tissue, as measured over soft muscle and skin.[6] It is mainly a mass reactance, increasing with frequency. We see that this impedance is

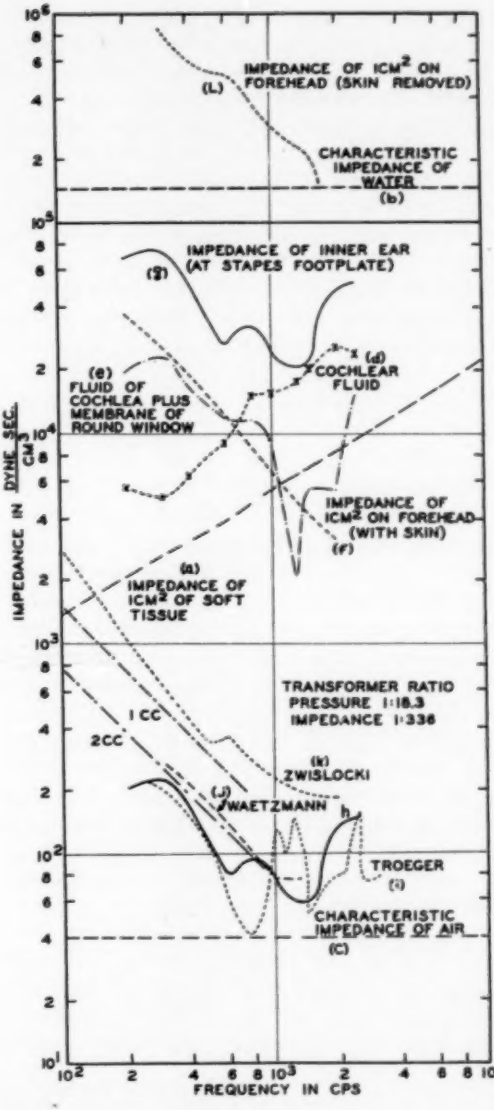


Fig. 1. Impedance Values Measured at Various Locations in the Ear and on the Body Surface—a. Impedance of area (1 cm²) overlaying soft tissue (upper arm); b. characteristic impedance of water; c. characteristic impedance of air; d. impedance of cochlear fluid; e. impedance of cochlear fluid plus membrane of round window; f. impedance (area 1 cm²) of the forehead (with skin); g. impedance of the inner ear measured at the footplate of the stapes; h. inner ear impedance as appearing at the tympanic membrane (curve g transformed by the middle ear); i.-j.-k. impedance values measured at the tympanic membrane; l. impedance measured on the forehead without skin (area 1 cm²).

considerably below the impedance for plane compressional waves in water, Curve *b*, which is so often cited as the approximate impedance of tissue to which the middle ear transformer has to match the impedance of the air, Curve *c*. The reasons for the difference between Curves *a* and *b* are not so much the differences in the properties of tissue and water, but they are the boundaries of the medium and the type of wave propagation set up in it. Over the whole audio-frequency range we hardly ever find compression waves in the body tissue; we always have shear waves and surface waves, which have different propagation velocity, absorption and wave length.[6] I think this fact cannot be stressed enough in discussing propagation of sound waves through tissue. Let us now look at the impedance of the cochlear fluid alone, Curve *d*, as measured by von Békésy.[5] We see this impedance is quite similar to the impedance of soft tissue. If we add to this fluid the elastic membrane of the round window we observe on the resulting impedance, Curve *e*, the following: For the lower frequencies the elastic component determines the impedance,* and this impedance is very similar to the impedance of other bony structures of the body. As an example, the impedance measured on the forehead is plotted as Curve *f*[1,2]; so we see the impedance values of the cochlea are of the same order of magnitude as the impedance of other body tissues. The impedance measured at the footplate of the stapes, given by Curve *g*, was obtained by von Békésy.[5] If we now multiply this impedance with the square of the pressure transformation ratio of the middle ear, as discussed by Dr. Lawrence, we obtain, as the impedance at the tympanic membrane much lower values of the impedance, Curve *h*. These impedance values, calculated from measurements on human cadavers, agree very nicely with the impedance measurements (Curves *i*, *j*, *k*) made *in vivo* at the tympanic membrane by several investigators.[4,8,9] Only at low frequencies is the impedance measured at the tympanic membrane determined by the impedance of the tympanic cavity which exceeds the transformed impedance of the inner ear, Curve *h*. Depending upon the

*Some of the statements made here about the phases of the impedance, i.e., if they behave like masses or elasticities, are not direct conclusions from Fig. 1, but follow from the original investigations.

volume of this cavity, we measure impedances of the general shape indicated in Fig. 1 by the impedances for air volumes of one and two cubic centimeters. Only here in this region, where the middle ear increases the transformed inner ear impedance, the middle ear transformer does not act as an "ideal transformer."

This elasticity of the middle ear cavity, together with the elasticity of the tympanic membrane itself are the reasons why the impedance measurements at the tympanic membrane have only limited value for diagnostic purposes; not all acoustic energy received by the tympanic membrane is conducted to the labyrinth. On the other hand impedance measurements are almost the only objective examination method available to make a real measurement of the sound conduction mechanism *in vivo*. It is regrettable that so far this method has never been refined and improved, so that it could be clinically applied to those cases for which its usefulness has been proven in principle: the diagnosis of conduction impairments.[3]

On Fig. 2 the absorption co-efficients resulting from the impedance values of Fig. 1 are shown. The absorption co-efficient indicates the percentage of the incident airborne sound energy that is absorbed by the tympanic membrane (Curves *a*, *b*, *c*) and conducted to the inner ear; or absorbed by soft muscle tissue (for example upper arm, Curve *d*) or bone (Curve *e*). Curve *f* indicates how little energy of a plane airborne sound wave is transmitted through a boundary between air and water. It should be noted here that in many cases, as Metz[3] has shown, conduction impairments in persons having normal tympanic membranes are detectable by a decrease of the absorption co-efficient measured at the tympanic membrane.

The absorption co-efficient shown in Fig. 2 for bony structure, Curve *e*, can hardly be used to calculate the threshold of bone conduction. It gives only the energy absorbed by the surface of the forehead, and does not tell us how much this energy is attenuated in the bone on its way to the inner ear. Most of this energy is already absorbed in the skin and not transmitted to the bone itself; therefore, whereas the energy absorbed at the tympanic membrane, Curves *a*, *b*, *c*, is, at

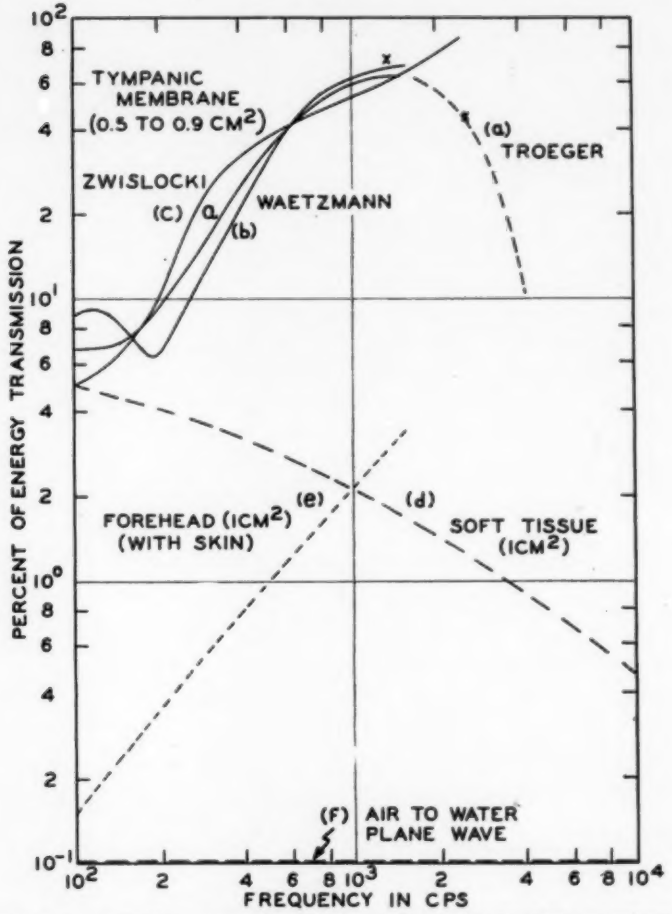


Fig. 2. Acoustic energy transmission through the tympanic membrane and other human body surfaces in per cent of incident sound energy.

least at the higher frequencies, almost completely transmitted to the cochlea, roughly only 1/1000 or less of the energy received by the forehead reaches the inner ear. This explains why in a free sound field our threshold for airborne sound, received by body areas other than the ears and transmitted

to the inner ear by bone conduction, is approximately 50 to 60 db below the air conduction threshold. In Fig. 3 some hearing threshold measurements are shown when the sound stimulus was exciting different areas of the body.[7] The data are plotted with reference to the air conduction threshold. It is remarkable how relatively little difference one observes in stimulating different areas of the body. This indicates that the main loss occurs at the transition surface.

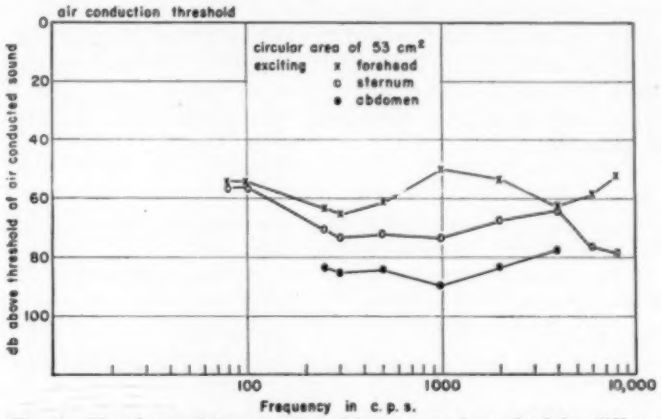


Fig. 3. Threshold of hearing for airborne sound received by different areas of the body relative to the air conduction threshold.

I do not want to go deeper into the problem of bone conduction here, and I think it was no accident that Dr. Lawrence omitted details on it. It is actually deplorable how little exact physical information we have about the bone conduction pathways in the tissue and about the details of how the bone conducted sound acts on the cochlea. Since it has been shown that impedance and skull resonances, measured on skull preparations, can be related reasonably well to *in vivo* measurements, such measurements should be possible with electroacoustic methods and tools.

The physical description of the mechanical conduction mechanism is not solved until we can describe the stimulus delivered to the electro-mechanical transducers. Von Békésy's[5]

observations, and his measurements of the mechanical constants of the cochlea, gave us the physical understanding and led to the theories describing the mechanics of the cochlea in general terms. Finer, but also important, details like the location and cause of nonlinear distortions and the mechanical energy finally transmitted to the hair cells are still very far from a solution. It looks as though a new tool must be found to obtain access to these dimensions, and this tool might well be the electrical activity of the inner ear itself. Dr. Lawrence's discussion of the distortion problem and the investigations we are looking forward to hearing about from Dr. Davis and his co-workers, indicate that further understanding of the electrical potentials might give us the tools to make another step ahead in measuring the mechanical conduction mechanism.

REFERENCES.

1. FRANKE, E. K.: The Response of the Human Skull to Mechanical Vibrations. WADC TR. 54-24, November, 1954.

2. FRANKE, E. K.: Response of the Human Skull to Mechanical Vibrations. *Jour. Acoust. Soc. Amer.*, 28:1277, 1956.

3. METZ, OTTO: The Acoustic Impedance Measured on Normal and Pathological Ears. *Acta Oto-Laryngol.*, Supp. 63, 1946.

4. TROEGER, J.: Die Schallaufnahme Durch das Aeussere Ohr. *Phys. Zeits.*, 31:26, 1930.

5. VON BÉKÉSY, G.: The Vibration of the Cochlear Partition in Anatomical Preparations and in Models of the Inner Ear. *Jour. Acoust. Soc. Amer.*, 21:233, 1949.

6. VON GIERKE, H. E., ET AL.: Physics of Vibrations in Living Tissues. *Jour. Appl. Physiol.*, 4:886, 1952.

7. VON GIERKE, H. E.: Personal Protection. *Noise Control*, 2:37, 1956.

8. WAETZMANN, E., and KEIBS, L.: Hoerschwellenbestimmungen mit dem Thermophon und Messungen am Trommelfell. *Ann. d. Phys.*, 26:141, 1935.

9. ZWISLOCKI, J.: Some Measurements of the Impedance of the Eardrum. *Jour. Acoust. Soc. Amer.*, 29:349, 1957.

SOME CLINICAL FEATURES OF SOUND CONDUCTION.

TERENCE CAWTHORNE,

London, Eng.

The sound conducting part of the auditory mechanism extends from the side of the head to the basilar membrane

which supports the organ of Corti. Any hindrance to the passage of sound waves down the external auditory meatus, across the ossicular chain of the middle ear cleft to the oval window, and finally through the labyrinthine fluids to the basilar membrane, will cause an impairment of hearing even though the organ of Corti and VIIIth nerve are normal; furthermore, the degree of deafness is a measure of the effectiveness of the block which may be such that air borne sound waves are prevented from reaching the organ of Corti.

A simple plug of cerumen in the external auditory meatus, particularly if it hinders the free excursion of the tympanic membrane, deserves pride of place as the commonest cause of conductive deafness. An effusion into the tympanic cavity, a vacuum within the middle ear space due to Eustachian insufficiency, or solid matter in the shape of adhesions or growth may also impede the transmission of sound, but the most effective, and next to cerumen the most common cause of conductive deafness, is found when there is an interference with the normal mechanism whereby sound waves enter and leave the labyrinth by means of the oval and round windows. In the Eighteenth Century Valsalva, Morgagni and Meckel all recognized stapes ankylosis as a cause of deafness, and late in the Nineteenth Century Politzer gave it the name otosclerosis.

More recently it has been appreciated that any interference with the normal phase relationship between the oval and round windows may affect the transmission of sound to the inner ear. This may happen if the ear drum is perforated so that the incoming sound waves fall more or less simultaneously on the two windows, thus cancelling each other out. The reason why the earlier operations for the relief of deafness due to otosclerosis by means of an opening into the lateral semi-circular canal were so rarely rewarded by success is that the operated area was closed up so that incoming sound waves fell with more or less equal and simultaneous pressure, both on the round window and on the new window in the lateral semi-circular canal.

Sourdille was the first to devise an open operation so that the incoming sound waves were free to pass through the new

window in the lateral canal, while the round window was shielded by the tympanic membrane. Sourdille's multi-stage procedure was simplified and improved by Lempert, whose fenestration operation has become the standard surgical procedure for the relief of deafness due to otosclerosis. Once the sound waves have entered the labyrinth they may be hindered by abnormal conditions of the fluid therein. It has been suggested that the variable low tone deafness in Meniere's disease is due to the increased pressure of endolymph interfering with the proper excursion of the basilar membrane.

One of the problems facing the aural surgeon when an otosclerotic patient presents himself hopefully as a candidate for fenestration, is to estimate the chances of success. Obviously if the organ of Corti itself is also defective, no operation to enable sound to re-enter the labyrinth, no matter how well it is carried out, will help if the organ of hearing is incompetent. In consequence surgeons have been chary of operating on any patients who exhibit any of the classical signs of perceptive deafness.

Now, as the movement of the stapes footplate in the oval window in response to sound becomes more limited as the otosclerotic bone spreads, a time must surely arrive when it will move hardly at all, even in response to powerful acoustic pressures. The effect of this will be to prevent the transmission of sound waves through the oval window. With only one window working but little effective sound is likely to reach the end-organ of hearing, and in consequence signs of cochlear insufficiency will be added to those of defective transmission, even though the cochlear end-organ is intact and ready to respond to any sound which may be permitted to reach it. Most of us have been brought up to believe that in advanced otosclerosis cochlear changes take place. Stacy Guild has shown that in the temporal bones of advanced otosclerotic deafness which he examined, there were no more histological changes in the organ of Corti and VIIIth nerve than could be accounted for by age; therefore, it seems to me that we must revise our view of advanced otosclerosis and regard the profound deafness as being due to inability of sound to reach an otherwise normal organ of hearing rather

than to irreversible degenerative changes in the cochlea or VIIIth nerve.

In support of this view it may be mentioned that in many of these patients with so-called "mixed deafness," the response to speech, providing that it is loud enough, is the same as in uncomplicated conductive deafness; whereas we know that in most cases of end-organ or nerve deafness the response to speech is quite different. I do not believe that in advanced otosclerosis the organ of Corti or the cochlear nerve are, because of certain conventional tests, necessarily at fault. The acid test in all these advanced cases is whether they are able to understand amplified speech. So long as they can do this there is reason to hope that the organ of Corti will respond to any sounds that are able to reach it.

Now for another aspect of the conduction of sound waves, this time within the labyrinth. I refer to the Tullio phenomenon, which relies for its effect upon a vestibular response to an acoustic stimulus. Tullio originally produced this effect by making an opening into the lateral semi-circular canal of pigeons, and then observing that in response to a loud sound there was a typical vestibular response of the eyes and the head.

Just before the War, I made an opening into the semi-circular canal of a patient with Meniere's disease. The stapes was free to move, and the labyrinthine structures were intact. The following short extract of a film taken in 1939 shows what happened. The small light attached to the earphone lights up when a sound of 80 db is on. It will be noticed that on the unoperated side nothing happens when the bulb lights up, but on the operated side it is followed by a deviation of the eyes and head. You may well ask, why does this not happen after every fenestration operation? The reason is that the stapes is not free to move. If it were, and I have seen it in patients who were submitted to a fenestration operation for perceptive deafness when the stapes was mobile, then this distressing effect is seen—distressing because in several cases I have had to destroy the labyrinth in order to relieve the patient of troublesome vertigo.

One final word about the footplate of the stapes in otosclerosis. I have tested its mobility at operation using the dissecting microscope in many cases, and I have also inspected the round window niche.

In very advanced cases of otosclerosis with profound deafness, the footplate and the distal part of the crura may be literally buried in otosclerotic bone, and occasionally I have found the round window niche also to be blocked by otoscerotic bone. On the other hand, in patients whose deafness is not so severe there is often some movement in the stapes, and I have been able to observe movement of the round window in response to pressure on the stapes.

In the Wernher Research Unit on Deafness at King's College Hospital Dr. Littler and I have been trying to measure the impedance of the stapes in otosclerosis, and this work is still in progress.

Regarding the relatively new procedure called mobilization of the stapes, which has been recently re-introduced by Dr. Rosen of New York, this is arousing great interest everywhere, and there is no doubt that it works in a proportion of cases. My impression so far is that it is most likely to be successful when the hearing loss for pure tones does not exceed 50 db; and being such a slight procedure, it can be justifiably recommended when only one ear is affected. An important point is that if it is unsuccessful it does not in any way prejudice the chance of a subsequent fenestration operation. Though it is a minor operation from the patient's point of view it is far from easy, and I feel that those who practice it should also be prepared to go through with a fenestration operation subsequently, should it not succeed.

New and beautiful plastic operations for the repair of perforated ear drums, and for the restoration of the ossicular chain on ears damaged by former infection, reported by Zolliver and Wullstein, are attracting much attention; also the provision of an external meatus and middle ear for children born with aplasia of the sound conduction mechanism is now an established procedure.

Dr. Heinrich G. Kobrak, Wayne University School of Medicine, Detroit, Michigan, presented a film on sound conduction in the ear. Unfortunately, due to his unexpected death a description of the film is not available.

TRANSMISSION AND TRANSDUCTION IN THE COCHLEA.*

HALLOWELL DAVIS,

Central Institute for the Deaf,

St. Louis, Mo.

My portion of our symposium deals with the cochlea as a sense organ. The domain extends from the oval window to the internal auditory meatus. Dr. Lawrence has told you how sound is conducted to the inner ear, and Drs. Galambos, Tasaki, Neff and others will pick up the story of the nerve impulses that emerge from it.

My story breaks into two major parts: the first is *acoustic analysis*; the second is *neural excitation*. These follow one another logically in the sequence of transmission and then transduction. They also follow in the order in which they have commanded scientific interest in the past and in regard to the extent of our present knowledge and understanding. Each part is primarily concerned with one of the two major dimensions of the acoustic stimulus. Acoustic analysis is concerned with how information concerning *frequency* is "coded" for transmission. This has been the historic and almost exclusive subject of the "auditory theory" of psychologists, but personally, as a physiologist, I am now even more interested in how, regardless of frequency, the ear can encompass its tremendous *dynamic range*; how it can respond to movements of its tympanic membrane of sub-molecular amplitudes; whether there is a biological amplifier that stands between the movements and the nerve impulses, and, if so, how it operates.

*Prepared under Contract N6onr 272 (03) between Central Institute for the Deaf and the Office of Naval Research. Reproduction in whole or in part is permitted for any purpose of the United States Government.

Thanks to the monumental work of Prof. Georg von Békésy, who should be telling you in person his half of this story, we know quite definitely the gross pattern of movement of the cochlear partition. This is the now-famous traveling wave pattern, which many of you saw illustrated in Prof. Békésy's motion picture. Equally well established is the proposition that the extent of the traveling wave pattern along the cochlear partition, and the position of its maximum of displacement both vary with the frequency of the sound waves. Thus the cochlea does act as an acoustic analyzer. The apical end of the cochlear partition is moved and its sensory nerves are stimulated only by low frequency sounds, and those at the basal end near the round window are stimulated by all sounds (of moderate strength) whether low, medium or high in frequency. This is not the exclusive one-frequency-to-one-place pattern of the piano keyboard, but it is a definite *place principle* of cochlear action; and it is not a matter of speculation but of observation. Békésy has seen it hundreds of times in actual cochleas and in models, and we have verified it time and again with our electrical recordings.

At the same time the cochlea also operates, within limits, on the telephone or frequency principle. Nerve impulses tend to group in successive volleys that reproduce the frequency of the stimulating sound waves. Up to the natural limit set by the refractory period of the nerve fibers, by the variability of their latency of discharge, and by the temporal dispersion introduced by the traveling wave pattern of cochlear action, the nerve impulses carry information about frequency *directly*, according to the frequency principle. Wever postulated his volley principle of firing-in-turn as an accessory to the basic frequency principle, and was proved right experimentally by Galambos and again by Tasaki. They will give you some of their details of the story later.

I have alluded above to a major principle of nervous transmission: namely, the *nerve impulses are all-or-none.* Another is that *in the nerve axons they travel in insulated channels.* They are triggered by the stimulus, but their energy is provided by the fibers. Their maximum sustained frequency is two to three hundred impulses per second in a

single nerve fiber. The overall task of the cochlea is to match this many-channeled single-valued communication system to the single continuously-variable channel of the open air, and then to code the particular acoustic signals in terms of patterns of nerve impulses. (I hope that Dr. Licklider will tell you whether I have used Prof. Gabor's terms "matching" and "coding" correctly, and also how modern auditory theory combines the place principle, the frequency principle, and the all-or-none principle.)

Several secondary principles, or propositions of physiology and biophysics, can be added in describing cochlear actions. Some are old, some are new, but all have been greatly illuminated by recent anatomical studies or physiological experiments. A few of these I shall discuss with you to the extent that time permits. One of these will be the complexity of the fine movements of the cochlear partition. Békésy has visualized them and finds them far more complicated than we had previously assumed. Likewise the electrical responses are more varied and more complex. They include not only the familiar cochlear microphonic but also one and probably two DC responses to AC stimulation. The ear acts as an acoustic rectifier and integrator, and the "summating potentials" are the electrical signs of this action. The endolymph has a unique chemical composition, and the endolymphatic space is strongly charged electrically relative to its surrounding tissues; but we shall hear evidence tomorrow that these unusual properties are not dependent upon one another. We can now see anatomical details of hair cells, nerve endings and supporting cells to a new order of magnification with the electron microscope, but the secret of their mechano-electric transducing or amplifying action is not revealed by it. The innervation of the hair cells is a complicated network, with contacts of many cells to a fiber and several fibers to a cell. More recently the efferent character of the bundle of Rasmussen has become clear, and Dr. Galambos will tell you how it modifies the sensitivity of the auditory receptors. Here, in a feed-back control from the central nervous system, is a really new principle of cochlear action.

Enough of generalities and previews. Let us look rapidly

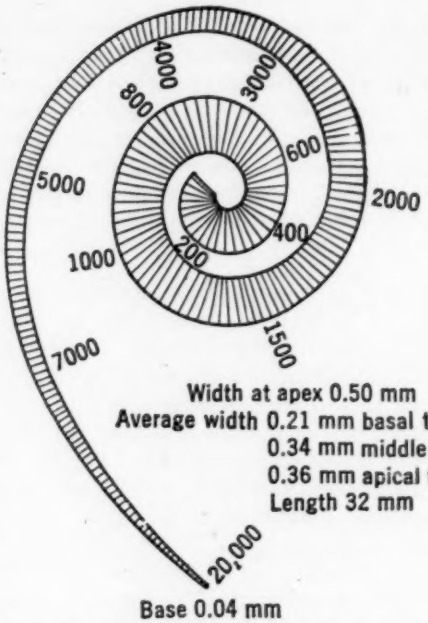


Fig. 1. Diagram of the human basilar membrane, showing the approximate location of the regions of maximum amplitude of movement for tones of different frequency. The width of the membrane is exaggerated relative to its length in order to show the widening more clearly (Stuhlman).

at some of the details. We are all audiologists, so I shall not take time to review the familiar basic anatomy of the cochlea; but in Dr. Békésy's absence I shall recall briefly the story of the traveling waves, and then pass on to some of the finer details.

MECHANICAL PROPERTIES OF INNER EAR STRUCTURES.

In the inner ear the basilar membrane widens gradually from 0.04 mm. at the stapes to 0.5 mm. at the helicotrema. Certain other measurements, such as cross section of the cochlear canal and relative sizes of certain types of cell in the organ of Corti, are also graded from end to end, but the important variation that allows the cochlea to act as a mechanical acoustic analyzer is in the width of the basilar mem-

brane. As a result of this variation, the stiffness ("volume elasticity") of the cochlear partition varies by a factor of at least 100 from one end to the other.

The cochlear partition has significant stiffness and also mass. Contrary to earlier opinions, it is not under tension. When cut, the edges do not retract. The movements of the partition, like those of the middle ear, are quite heavily, but not critically, damped. Because of the gradation in stiffness and mass, different parts of the basilar membrane have different resonant frequencies, but the various parts cannot move as independent resonators. The basilar membrane, and the organ of Corti on it, are continuous structures. Their elements are coupled to one another elastically and also by friction. The endolymph and the perilymph provide some of the friction.

TRAVELING WAVE PATTERN OF THE COCHLEAR PARTITION.

An increase of pressure on the footplate of the stapes, caused by a sound wave, causes a wave of acoustic pressure to spread through the fluid in the cochlea with approximately the speed of acoustic transmission in water, about 1500 m/sec. Because of the small size of the cochlea, the increase in pressure is virtually simultaneous throughout. The only elastic portion of the walls of the bony labyrinth is the round window, and its membrane bulges outward and allows inward movement of the stapes. Fluid moves from oval window toward round window. The cochlear partition lies in the path of this movement, and it bulges toward the round window. If the movement is very slow, there will also be some flow through the helicotrema, but all parts of the cochlear partition do not move with equal promptness. The relatively stiff portion in the basal turn moves very nearly in phase with the driving force, but the more flexible apical portions, particularly those whose resonant frequency is lower than the frequency of the acoustic wave that is driving the partition, tend to lag behind. As the acoustic wave reverses its pressure, the portion that is "tuned" to lower frequencies tends to overshoot and continues to lag behind the driving force exerted on it by the acoustic pressure in the fluid. Thus, because of

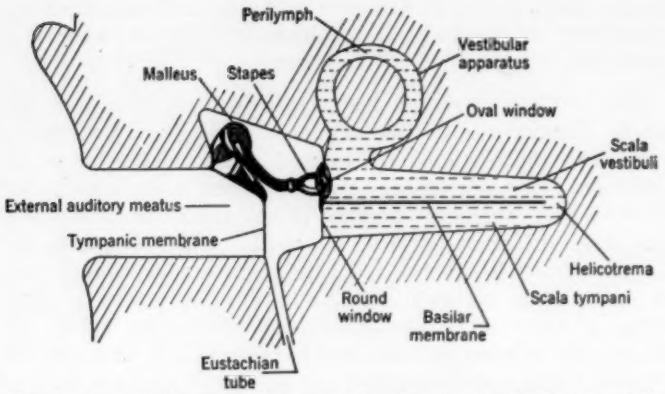


Fig. 2. Overall diagram of the ear, showing the ossicles in the middle ear (the incus is not labeled) and the fluid-filled inner ear. The cochlea is drawn as a short, straight chamber instead of a long, coiled canal (Bekesy).

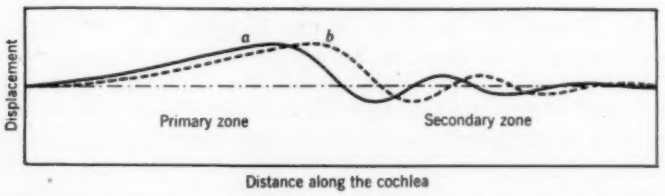


Fig. 3. The solid line (a) shows the displacement pattern of the cochlear partition at one instant and the broken line (b) the pattern an instant later. Actually the waves die out quite rapidly in the secondary zone, and their wave length becomes progressively shorter and shorter (Wever, Lawrence and Bekesy).

the gradation of stiffness, a *traveling wave* of displacement appears on the cochlear partition (see Fig. 3); furthermore, because of the continuity of the partition, the stiffer portion, moving almost as a unit, drives the more flexible portion.

The traveling wave increases in amplitude as it moves apically and reaches its maximum near the region where resonant frequency of the basilar membrane corresponds to the frequency of the driving waves (see Fig. 4). The amplitude of movements falls off rather rapidly beyond this point. The phase lag increases more and more rapidly as the travel-

ing wave moves on toward the apex. The velocity of travel, therefore, diminishes, and consequently the wave length of the displacement pattern becomes shorter. A little distance beyond the position of maximum amplitude there is no significant movement at all. In the region of rapid diminution of amplitude the phase lag amounts to a full cycle or more.

If the driving frequency is increased, the position of maximum amplitude moves toward the oval window; if it is decreased the maximum moves toward the apex (see Fig. 5). At about 100 c.p.s. it is very close to the helicotrema. At

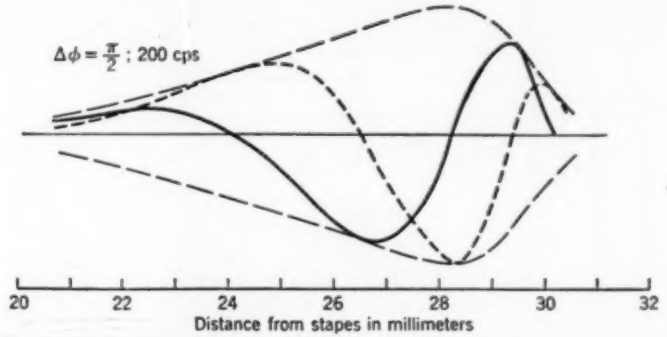


Fig. 4. Two displacement patterns are shown at an interval of a quarter of a cycle. The dashed line is the envelope of the instantaneous patterns and corresponds to the dashed curve in Fig. 5, upper (Bekesy).

2000 c.p.s. there is very little movement beyond the mid point of the cochlear partition; but the extreme basal end of the partition moves in response to *all* frequencies within the audible range.

The unsymmetrical traveling wave pattern of movement, with its rather flat maximum of amplitude and its abrupt apical reduction in activity, has been shown to be a necessary and predictable consequence of the principles of acoustic resonance in a system such as the cochlea with gradation of stiffness, mass, damping and coupling. The traveling wave pattern has been reproduced in appropriate physical models, and it has been observed directly in the cochlea under the

microscope with stroboscopic illumination. It allows the cochlea to act as a mechanical frequency analyzer, because the extent of activity and position of maxima vary as functions of frequency. It introduces additional features, such as asymmetry, progressive time and phase lag, and significant longitudinal as well as transverse bending of the cochlear partition, that contribute to the pattern of neural excitation that results from the movements of the partition.

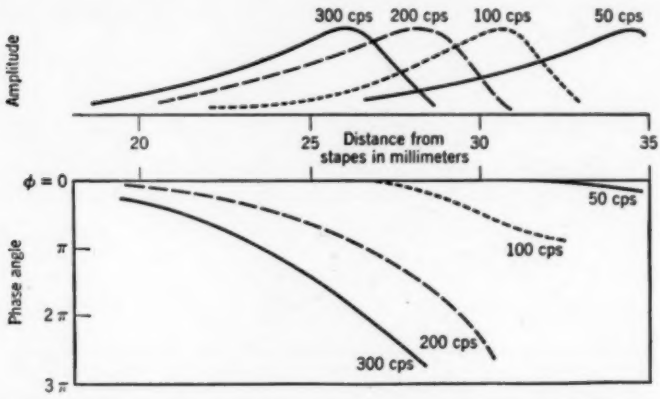


Fig. 5. The upper set of curves shows the amplitude of displacement of the cochlear partition at various distances from the stapes, and for several driving frequencies. The lower set of curves shows the corresponding phase angles (Bekesy).

THE FINE MOVEMENTS OF THE ORGAN OF CORTI.

The fine movements of the organ of Corti and the tectorial membrane have been observed under the microscope by stroboscopic illumination and described in some detail by Békésy. In any one segment the basilar membrane, organ of Corti, tectorial membrane, and usually Reissner's membrane also, move in phase with one another. The basilar membrane is fibrous and elastic, and basically it determines the traveling wave pattern of vibration described above. The cells of Hensen form a soft cushion supporting the stiffer plate of the reticular lamina. The tectorial membrane is hinged like the cover of a book along the edge of the limbus. It is composed

of a system of diagonal fibers, and also a jelly-like substance. It yields to slow movements but is quite stiff and resistant to quick movements. It returns rather slowly after being detached from its normal attachment to the organ of Corti and then displaced.

Apparently, as the basilar membrane bulges "upward" or "downward" (see Fig. 7), the stiff reticular lamina tends to

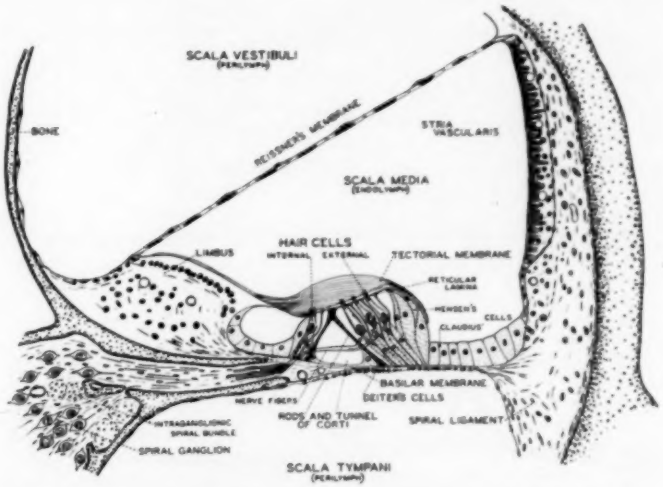


Fig. 6. Camera lucida drawing of a cross section of the cochlear partition in the second turn of a guinea pig cochlea. The attachment shown here of the tectorial membrane to the inner supporting cell, and to Hensen's cells, is based on microdissection of fresh, unfixed specimens.

rock, on the support of the rods of Corti, around an axis at the attachment of the basilar membrane to the bony modiolus. The tectorial membrane swings on its attachment to the limbus. The result is a shearing action between the tectorial membrane and the reticular lamina (see Fig. 8). The "hairs" arise from the cuticular plates of the hair cells, which are set firmly in the reticular lamina, and their outer ends are firmly imbedded in the tectorial membrane; therefore, as the basilar membrane bulges, the hairs are bent. The force of the move-

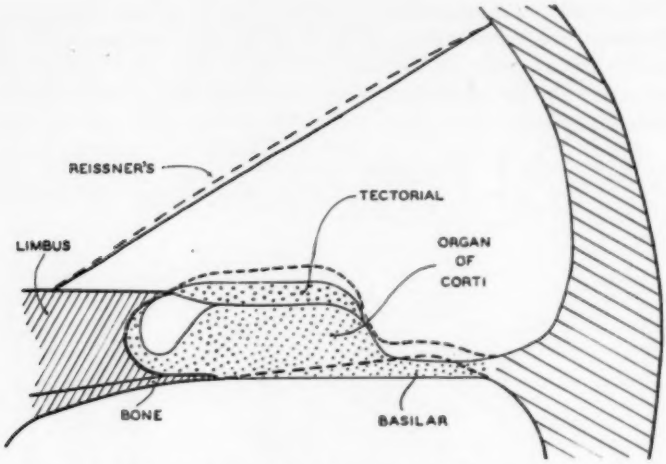


Fig. 7. Probable pattern of displacement of the cochlear partition seen in cross section.

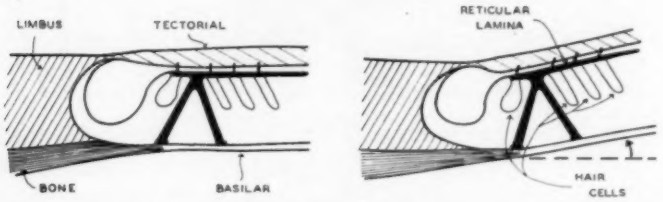


Fig. 8. Probable pattern of shearing action between tectorial membrane and reticular lamina, with bending of the hairs.

ments of the cochlear partition is rather efficiently concentrated on the shearing action.

The movement described above is associated with an approximately *radial* displacement of Hensen's cells, as seen under the microscope, and a corresponding radial or slightly diagonal bending of the hairs. This movement is characteristic on the basal side of the position of maximal amplitude. On the apical side, however, due to the shorter wave length of the traveling wave and sharper longitudinal bending of the basilar membrane, a *longitudinal* movement predominates,

and the hairs are presumably bent longitudinally instead of radially (see Fig. 9).

The exact significance of these different directions of movement in relation to the excitation of nerve impulses by the hair cells is still a matter of speculation, but the bending of the hairs is the final and critical mechanical event that

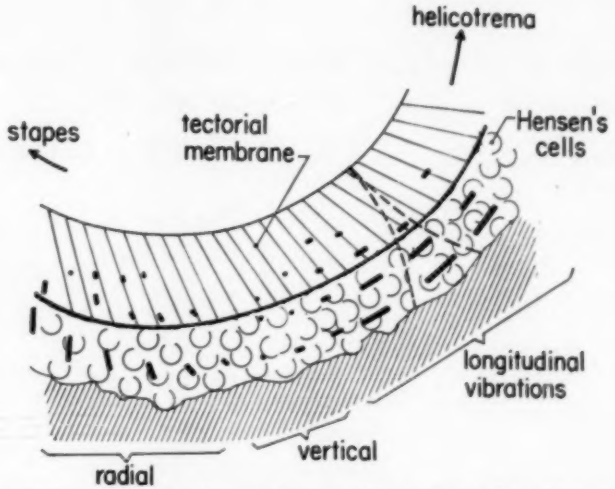


Fig. 9. Movements in the cochlear partition, viewed from the upper edge of the stria vascularis. The greatest amplitude of displacement in the pattern shown in Fig. 6 is in the region here labeled "vertical" (Bekesy).

has been recognized in the mechanism of stimulation. At this point the significant events apparently become electrical, for this bending of the hairs seems to release energy in the form of biolectric potentials, and these potentials are in all probability an important intermediate step in the mechanism of excitation of the auditory nerve fibers.

The *endolymph*, which fills scala media, differs sharply from perilymph in its ionic content. Unlike all other body fluids, it is high in potassium and low in sodium. It more nearly resembles intra-cellular fluid in this respect. Typical

analyses of endolymph, perilymph and cerebro-spinal fluid are given in Fig. 10. The endolymph has sometimes been described as "viscous," but this is probably true only for certain fish and perhaps other lower forms.

The endolymph is probably secreted by the stria vascularis. Whether it is also reabsorbed wholly or only in part by the same structure is a matter of debate.

	Spinal Fluid	Perilymph	Endolymph
	milliequivalents per liter		
Potassium	4.2	4.8	144.4
Sodium	152.0	150.3	15.8
Chloride	122.4	121.5	107.1
	milligrams per cent		
Protein	21.0	50.0	15.0

Fig. 10.

INTRACELLULAR POTENTIALS.

Nearly all cells show a negative intracellular potential. Explorations of the cochlea and the auditory nerve with very fine microelectrodes reveal these intracellular potentials, ranging from –60 or even –80 mv. relative to the potential of the perilymph in large cells, such as Hensen's or Claudius' down to –20 mv. or so in the cells of Reissner's membrane. The exact value seems to be a function of the amount of injury caused by the microelectrode; the greater the injury the lower the value. The hair cells, like the nerve fibers and supporting cells, are electrically *negative* internally.

THE ENDOCOCHLEAR POTENTIAL.

The interior of scala media, the endolymph, is electrically *positive* relative to the perilymph in scala vestibuli and scala tympani and to the spiral ligament and extra-cochlear tissues in general. This potential is +80 mv. It is encountered abruptly at the point where the exploring electrode enters the endolymphatic space. The change of potential in going

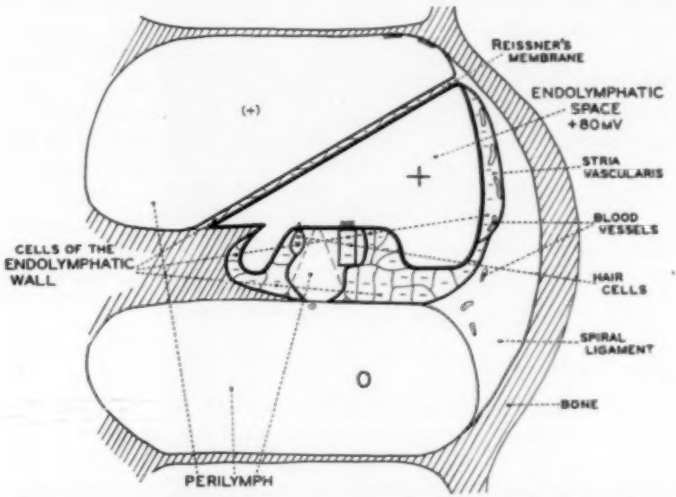


Fig. 11. The endolymphatic space, bounded by the heavy line, carries a positive electrical charge of 80 mv. relative to the perilymph in scala tympani. Scala vestibuli is not more than 5 mv. positive. The interiors of all of the cells of the cochlear partition are electrically negative.

from the interior of a hair cell through its cuticular layer into scala media is thus about 150 mv.

The endocochlear potential is closely dependent on an adequate oxygen supply. It falls, reversibly, to a very low level at the stage of asphyxia that is reached in extreme Cheyne-Stokes respiration (in anesthetized, moribund guinea pigs). Full recovery requires only a few seconds after a large single gasping inspiration. It is also abolished rapidly by injection of cyanide or azide into scala tympani or scala media. It is

not immediately affected by injection of isotonic potassium chloride, choline chloride, or potassium glutamate into scala tympani or scala vestibuli. It does fail, although less rapidly than with cyanide, following surgical injury to scala media or the injection into scala media of a solution that differs substantially in ionic content from the analytic figures for endolymph given in Fig. 10.

The endocochlear potential is modified by displacement of structures within the cochlear partition. Displacement of the basilar membrane toward scala tympani, as by injection of fluid into scala media or an inward movement of the stapes, causes an increase in the positive potential by as much as 5 or 10 mv. Movement in the opposite direction, as by outward movement of the stapes, causes even greater reductions in the potential. Movements of Reissner's membrane alone are not effective, but movements of tectorial membrane relative to the reticular lamina, when it is manipulated by a microneedle, produce just such changes in potential. The changes are related to displacement, not to velocity, and are sustained as long as the displacement is maintained.

The *source* of the endocochlear potential has not been identified, although both hair cells and the stria vascularis have been suggested. The *changes* in endocochlear potential, described above, are clearly associated with the organ of Corti, almost surely the hair cells, but it is not certain that the resting positive endocochlear potential is generated here. Perhaps a separate electric response to mechanical stimulation occurs in the hair cells and simply adds to the background potential that is produced by another "generator".

THE COCHLEAR MICROPHONIC AND THE SUMMATING POTENTIALS.

The cochlear microphonic and two summating potentials (positive and negative) are all electric responses to acoustic stimulation. The cochlear microphonic is linearly proportional, up to a limit, to the displacement of the cochlear partition and thus, indirectly, to the instantaneous acoustic pressure. The microphonic thus reproduces the *wave form* of the acoustic stimulus (see Fig. 12). The summating potentials are proportional, not to any instantaneous value of the acoustic

signal, but to a root-mean-square value, integrated over a very short time. Thus the summating potentials reproduce approximately the form of the *envelope* of the original acoustic signal. The positive and the negative summating potentials are opposite in sign. They can be separated by the greater vulnerability of the positive summating potential to oxygen

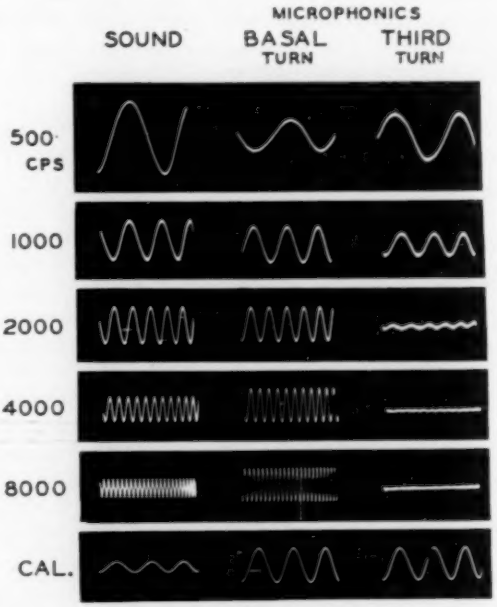


Fig. 12. The cochlear microphonic, recorded by intracochlear electrodes on opposite sides of the cochlear partition, reproduces the sine wave form of a pure tone. The basal turn responds to all frequencies, but the third turn responds only to frequencies below about 3000 c.p.s.

lack and other injury, and by the more apical site of generation of the negative response. The input-output relations of the summating potentials are now being investigated. They are not simple.

The cochlear microphonic is generated at the cuticular surface of the hair cells. This is clearly proved by exploration

with microelectrodes. The microphonic, and in all probability the summating potentials also, seem to reflect the bending of hairs in the appropriate direction. Apparently at intensities high enough to evoke the summating potentials, some kind of mechanical rectifying or detector action takes place in the inner ear to cause an unsymmetrical, persistent one-way bend in the hairs of certain cells. In some cases the bending is probably across, in others lengthwise of the organ of Corti. (A theory that includes this and several other aspects of the electrophysiology of the cochlea has recently been published in *Physiological Reviews*, 1957.)

Both the cochlear microphonic and the summating potentials are continuously graded responses, linearly related within limits, to the intensity of the acoustic stimulus, and with no true "threshold" like that of all-or-none action potentials. No evidence of any all-or-none response in the sensory cells or of a refractory period has been found, even when the cochlear microphonic was recorded from an electrode inside a hair cell. Both the microphonic and the summating potentials show very little or no fatigue or adaptation.

The cochlear microphonic "appears," in the sense that it reaches an rms. value of a microvolt or thereabouts, at a much lower sound pressure level than the summating potential, except at the extreme high-frequency limit of response. In the basal turn in the guinea pig the increase is linear, with the sound pressure level up to about 80 db. relative to 0.0002 microbar. The response then increases more slowly and usually goes through a maximum. At low frequencies ordinary mechanical non-linear distortion limits the output. At higher frequencies the electrical output is limited, but the wave form of the cochlear microphonic is well maintained, with little or no "peak-clipping," in contrast to the flat-topped waves seen at lower frequencies. The summating potentials "appear" some 20 db. above the cochlear microphonic, and continue to increase up to limits set only by acoustic trauma to the organ of Corti.

The cochlear microphonic, like the endocochlear potential, is closely dependent on an adequate oxygen supply. Later

we shall hear evidence that the cochlear microphonic is not entirely dependent on the positive potential of the endolymph. The cochlear microphonic, however, apparently does represent, as Békésy originally implied, an amplifier action in which the acoustic stimulus "valves" the energy from a pre-existing "biological pool," such as the endocochlear potential.

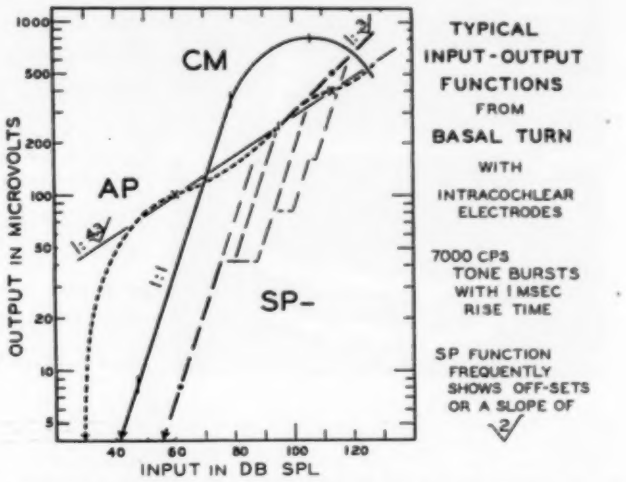


Fig. 13. The voltage of the cochlear microphonic is proportional to the sound pressure level up to about 80 db. SPL, but goes through a maximum at about 105 db. SPL. The height of the action potential spike (N_1) increases rapidly from threshold, and then gradually at a rate that varies between the third and the fifth root of the sound pressure level. The negative summating potential is directly proportional to sound pressure level when it first appears. It does not show a maximum like CM, but its increase is often interrupted by plateaus. No single typical curve can yet be drawn for SP.

We may add that the negative intracellular polarization is also such a "pool." In any case it now seems clear that the sustained changes in the endocochlear potential noted above certainly represent a modulation of a biological source of energy, and not simply a passive physical transducer mechanism.

The summating potential, as usually seen, is a displacement of the baseline of the oscilloscopic record on which the cochlear

microphonic is superimposed. It is a paradoxical fact that, with mild anoxia, abnormal ionic concentrations, etc., while the cochlear microphonic diminishes the negative summating potential increases. The diphasic effect of anoxia, etc., is best explained by assuming that, *1.* there is a positive as well as a negative summating potential, generated by a different

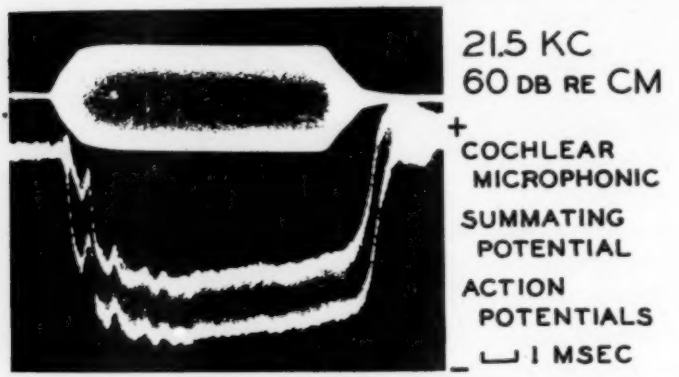


Fig. 14. The upper trace is the electrical pattern of a tone burst at 21.5 kc. An electrode on the round window of guinea pig records a complex electrical response consisting of cochlear microphonic (at the frequency of the sound waves), summating potential (downward displacement of the base line), and action potentials (the peaks and notches on the descending limb of the SP response). The stimulus is maximal for CM. Its sound pressure level is 60 db. above the level needed for a CM response of 3 μv. The apparent decay of SP during the tone burst is an artifact due to the condenser coupling of the recording amplifier. After the end of the tone burst the CM response to the reverberation continues to be nearly maximal.

set of sensory cells; and that *2.* the positive response is more sensitive to anoxia than the negative. The negative summating potential seems to be stronger, although higher in threshold, and only under more severe anoxia or ionic injury does it in its turn weaken and finally disappear.

We shall rely on Drs. Galambos and Tasaki to provide further details concerning the nerve action potentials in the second order and first order auditory neurons respectively. Let us devote the remaining moments to a quick interpretive summary of some of our statements about cochlear mechanics,

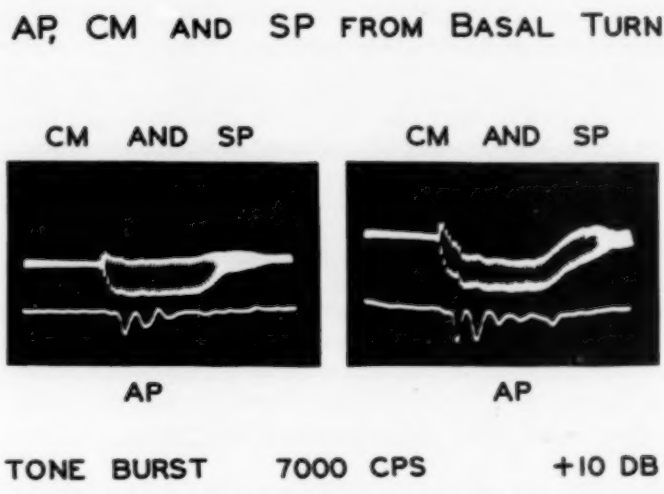


Fig. 15. The AP response can be separated from CM and SP by using intracochlear electrodes, an appropriate input network and two recording channels. The stimulus for the responses at the right was 10 db. stronger than for those on the left. Note that AP and SP- have increased, while CM has decreased slightly.

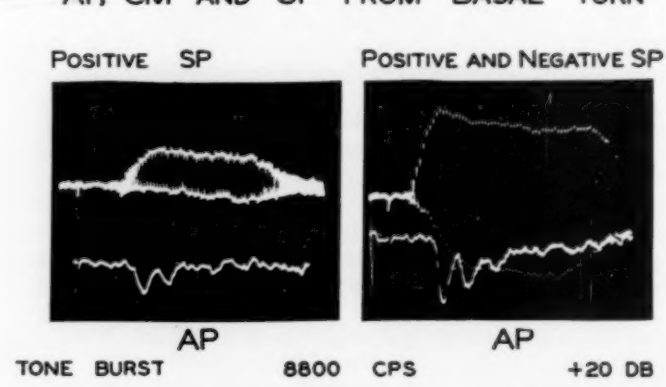


Fig. 16. In some preparations, with weak stimuli, the SP response is dominantly positive. A stronger stimulus gives a composite SP response.

and to the relations between movement and potential, and between potential and nerve impulses.

We conceive that, as the basilar membrane, and the organ of Corti with it, bulge one way or the other, there is a shearing action between the stiff reticular lamina of the organ of Corti and the stiff but viscous tectorial membrane that lies in contact with it, because the tectorial membrane pivots around a different axis, as illustrated in Fig. 7. The shearing action bends the hairs of the hair cells, which are attached both to the organ of Corti and to the tectorial membrane. This bending is the mechanical movement that is critical for stimulation. Protection against too great bending is probably provided by the attachment of the tectorial membrane directly to the outer and inner borders of the organ of Corti.

The longitudinal bending causes longitudinal vibratory movements among the cells of the organ of Corti, and presumably bends the hairs lengthwise of the cochlear partition. The longitudinal bending is sharpest in the "cut-off" region on the apical side of the maximum and is probably negligible on the basal side. It is uncertain as yet whether the external and the internal hair cells are equally sensitive to radial and to longitudinal bending of their hairs. Probably they differ, and the differential stimulation of the two sets by the different directions of bending allows possibilities, through inhibitory neural interactions within the central nervous system, of sharpening the "place" aspect of frequency discrimination.

The traveling waves are known to produce eddies in the cochlear fluids on the apical side of the position of maximal amplitude. The forces that produce eddies, we believe, also tend to cause an unsymmetrical longitudinal shift or "creep" to the tectorial membrane relative to the organ of Corti. Such a shift would cause a one-way longitudinal bending of the hairs. This would be a mechanical rectifying action. It allows the cochlea to "detect" efficiently and respond with nerve impulses to high-frequency acoustic signals above 2000 per second. Just as the cochlear microphonic is the electrical sign of a symmetrical vibratory bending of the hairs, we believe the negative summating potential is the electrical sign of an unsymmetrical, rectified longitudinal shift. This shift

is strongest on the apical side of the position of maximum excursion. The sustained bend of the hairs presumably acts as a steady stimulus to the hair cells that are affected, but compared to the alternating shearing movements revealed by the cochlear microphonic this mechanism is relatively insensitive. The rectifying action, as revealed by the negative summating potential, continues to increase, however, after the vibratory movements, and with them the cochlear microphonic, have reached their maximum. The rectifying action, however it is produced, seems to be a mechanism that significantly extends the dynamic range of the ear.

The complete theory, as presented elsewhere, considers the mechanism of limitation of crosswise bending (and with it the cochlear microphonic) in more detail and it also includes a second rectifying action, associated with the crosswise bending, that depends on the viscous properties of the tectorial membrane. This second mechanical rectifying action and consequent one-way bias of the hairs is invoked as the basis of the positive summating potential; but this extension of the theory, as well as a possible inhibitory action of the positive summating potential, is admittedly more speculative than the postulate of the longitudinal "shift" and its production of the negative summating potential.

The association of the cochlear microphonic with the bending of the hairs seems very well established. The mechanism that connects the two is completely obscure, however. A vague suggestion that the mechanical distortion changes the electrical resistance of the upper ends of the hair cells has been offered but without supporting evidence. Whatever the mechanism, the bending of the hairs is supposed to account for not only the cochlear microphonic but also both of the summating potentials, but these three electrical responses, it should be noted, are observed phenomena, not theories.

Consideration of the extreme sensitivity of the ear, and also the fact that a potential change persists indefinitely if a static displacement of tectorial membrane relative to the reticular lamina is maintained mechanically, leads to the conclusion already stated that *the energy of the electrical re-*

sponses is derived from the metabolism of the tissues, not from the acoustic stimulus. The latter serves merely to "valve" the flow of energy from the biological source. The result is an amplifier action in the sense organ prior to stimulation of the nerve fibers.

The cochlear microphonic and the negative summating potential are believed to excite directly the nerve fibers in

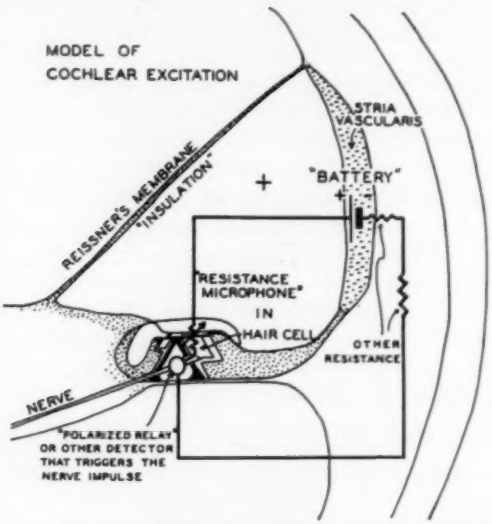


Fig. 17. An electrical model of excitation of nerve impulses in the cochlea. Additional "batteries", not shown in the diagram, are located at the cell membranes of the hair cells and of the nerve endings. The return circuit from nerve ending to the stria vascularis is not restricted to the narrow anatomical path indicated in the diagram, but is diffuse through all intervening tissues except the scala media.

contact with the hair cells. Only a passive role as electrical conductors is ascribed to the nerve endings. No latency can be seen between mechanical displacement of the cochlear partition and the cochlear microphonic. It is not yet clear whether there is synapse-like delay in excitation or whether the observed latency of action potentials (in the modiolus) should be attributed chiefly to conduction time in the non-

medullated dendritic branches of the primary neurons in the organ of Corti.

At frequencies below about 300 c.p.s. each sound wave acts like an individual stimulus to the nerve fibers. Excitation apparently occurs during the "falling phase" of the cochlear microphonic, *i.e.* while scala media (and vestibuli) is becoming more negative relative to scala tympani. This corresponds to the phase of *outward* movement of the stapes. The phase relation of neural excitation to the cochlear microphonic is correct for the electrical theory, although this may not be immediately obvious. The current flows from hair cell into nerve fiber and outward across the nerve membrane, and thus can excite the non-medullated dendritic terminals like one tremendous node of Ranvier, or like non-medullated fibers elsewhere. Spatial summation between the several hair cells attached to a given nerve fiber is clearly positive, and also a facilitating action between summating potential and cochlear microphonic.

This is at least a reasonable working hypothesis or "model" of the action of the cochlea as a sense organ.

ACKNOWLEDGEMENTS.

A more complete discussion of much of the material in this article, and a full list of references will be found in DAVIS, H.: "Biophysics and Physiology of the Inner Ear." *Physiol. Rev.*, 37:1-49, 1957.

Some pertinent additional material appears in another paper entitled "Modification of Cochlear Potentials Produced by Streptomycin Poisoning and by Extensive Venous Obstruction," by the present author and his associates, in this volume.

Several sections of this paper are taken almost verbatim from the chapter prepared by the present author for the forthcoming "Handbook of Physiology" of the American Physiological Society.

The courtesy of the following authors, journals and publishers in allowing reproduction of figures or data is gratefully acknowledged:

Fig. 1. STUHLMAN, O., JR.: "An Introduction to Biophysics." John Wiley & Sons, 1943.

Fig. 2., 4., 5. VON BÉKÉSY, G.: "Handbook of Experimental Psychology." John Wiley & Sons, 1951.

Fig. 3. WEVER, E. G.; LAWRENCE, M., and VON BÉKÉSY, G.: *Proc. Natl. Acad. Sci. U. S.*, 40:508, 1954.

Fig. 6. DAVIS, H.: *Jour Acoust. Soc. Amer.*, 25:1180, 1953.

Fig. 7., 8., 17. DAVIS, H.: "Physiological Triggers." American Physiological Society, 1956.

Fig. 9. VON BÉKÉSY, G.: *Jour. Acoust. Soc. Amer.*, 25:786, 1953.

Fig. 10. Data from SMITH, C. A.; LOWRY, O. H., and WU, M. L.: THE LARYNGOSCOPE, 64:141, 1954.

Fig. 11. TASAKI, I.; DAVIS, H., and ELDREDGE, D. H.: *Jour. Acoust. Soc. Amer.*, 26:765, 1954.

Fig. 14. PESTALOZZA, G., and DAVIS, H.: *Amer. Jour. Physiol.*, 185:595, 1956.

TRANSMISSION AND TRANSDUCTION IN THE COCHLEA.

Theoretical Discussion of Hallowell Davis' Paper.

J. C. R. LICKLIDER,*

Bolt Beranek and Newman Inc.

Cambridge, Mass.

My first comment on Dr. Davis' masterful job concerns the theoretical significance of Professor Békésy's experimental contributions: on the one hand, they displaced the speculations, most of which had been pretty far afield, about cochlear mechanics, and they sent the speculators scurrying up the auditory system to happier hunting grounds; on the other hand, they set the stage for a series of mathematical theories, not theories so much as assimilations, that reduced to formulas the essential schema of the cochlear mechanical frequency analysis.

It is probably impossible to overstate the importance, to the rest of auditory theory, of having this solid foundation on which to build. If one could ask for more without being

*Formerly at Massachusettes Institute of Technology.

greedy, he would ask for more direct observational data on the phase response of the cochlear partition, particularly at frequencies above 500 c.p.s.; but to have the picture we have of the mechanical analysis is so good that we should not be very greedy. Taking advantage of the established reasonableness of the assumption of linear superposition, one can, at least in principle, determine rather approximately, as a function of time, the pattern of displacement of the cochlear partition in response to any specified acoustic stimulus, sinusoidal or complex, periodic or aperiodic.

My second comment has to do with two different theoretical points of view from which we can examine the next stage of the auditory process. By the next stage, I mean the transduction from mechanical displacement of the cochlear partition to excitation of the neurons of the auditory nerve. The first point of view is that of the audiologist, or the experimental psychologist, who wants to understand the overall behavior of the individual. The other point of view is that of the physiologist who wants to know exactly what does what.

A few years ago, we had only a very tentative picture of this second stage of the auditory process. The displacement of the cochlear partition squeezed the hair cells; the hair cells shocked the nerve fibers, and impulses went up the VIIIth nerve. Today, however, the mechano-neural transformation is the focus of research attention. Does it play a role in the sharpening of the mechanical frequency analysis? To what extent are its parameters under the control of higher centers? Does the band width of the "cochlear filter" vary with the alertness of the organism, with the listeners' set, with the task assigned?

It is evident from Dr. Davis' summary that those questions do not yet have positive answers, but equally evident that answers are at least on the way.

The primary concern of many of the researchers who are hard at work on the mechano-neural transformation is an understanding of the physiology of the process. What structures respond in what manner under what conditions? The main interest of many audiologists and psychologists, on the

other hand, is an understanding of the form of the transformation. They are less interested in knowing what structures mediate it than they are in knowing the shape of the "transfer characteristic." Their hope, of course, is to apply the mechano-neural transformation to the signal that results from the mechanical cochlear analysis of the acoustic stimulus. That would carry the quantitative description of the process through another stage and give us a picture of the incoming signal as seen by the nervous system.

It seems to me worthwhile to distinguish between the advances in the two areas that correspond to the interests of the physiologist, on the one hand, and the audiologist or psychologist, on the other. Let us consider them in reverse order.

Insofar as the form of the transduction is concerned, the major advance, in what Dr. Davis has described, is the demonstration that the cochlear microphonic potential is supplemented by the summating potential. The microphonic normally is dominant at low frequencies and low intensities; the summating potential at high frequencies and high intensities. The summating potential appears as though it were derived through rectification and smoothing of the microphonic potential, and those two operations give us a rough picture of the form of the process. From the recent developments, we begin to see how the auditory system gets its extreme dynamic range. We see also that its behavior in response to strong signals is fundamentally different from its behavior in response to weak signals.

On the physiological side, the fundamental advance was the understanding that the cochlear potentials are energy-release phenomena and not direct-energy-transduction phenomena. Relating the physiology to the anatomy, there is the clear evidence that the focal point of the process is the hair cell.

When it comes down to the details of the process, however, the picture is extremely complicated. There are actually seven potentials to be considered, and there are at least seven structures that are regarded by one investigator or another as playing important roles. The opportunities for theory

construction, here, are, therefore, obviously quite as good as they ever were in the study of the mechanical action of the cochlea. I think that Dr. Davis has displayed both great courage and great skill in his discussion of "which structure mediates which functions." He has at every step, I think, picked the best and currently most plausible hypothesis. I am sure that he does not expect to be 100 per cent correct. I should not be surprised, however, if he touched 95 per cent, and to do that is a tremendous accomplishment.

TRANSMISSION AND TRANSDUCTION IN THE COCHLEA.

CLINICAL DISCUSSION.

JOHN R. LINDSAY,

University of Chicago,

Chicago, Illinois.

It is gratifying to the otologist to hear of the extensive research being carried on concerning the problems of hearing. Dr. Davis' presentation of evidence pertaining to the physiology of the hearing mechanism within the cochlea is of direct interest in the case of end-organ lesions, but does not lend itself readily to discussion from the clinical viewpoint. I believe it would be more appropriate for the otologist to call attention to a few of the clinical problems which are in urgent need of investigation.

The most common cause of serious hearing impairment in early adult life is the condition known as otosclerosis. The cause of otosclerosis is not known, nor is there any known means by which the progress of the disease can be inhibited. Although tremendous progress has been made in recent years in improving the hearing through surgery in certain selected cases of otosclerosis, there are many cases for which surgical procedures offer little or no help. There are many cases which, because of progressive nerve loss, cannot be helped either by medical or surgical means, and a few for whom the modern hearing aids are inadequate.

The cause of nerve degeneration associated with otosclerosis is not yet definitely known. Dr. Cawthorne has presented his view to you on this problem; yet on the basis of the histopathology in some advanced cases of otosclerosis there has been evidence of irritation of the inner ear and both degeneration of the sensory structures and a labyrinthitis ossificans.

Factual information is also needed to explain the severe deafness which sometimes follows relatively mild trauma to the head. Experimental work has furnished much information relative to acoustic trauma and the effect of high intensity noise on the inner ear; however, none of these experiments has adequately explained the profound deafness, sometimes unilateral and sometimes bilateral, which may result from striking the head on the pavement, for example, without causing unconsciousness and sometimes without evidence of fracture.

It appears from the clinical course that in this type of case the pathology differs from that produced experimentally by severe blows to the head. It is not explained on the basis of a transverse fracture of the pyramid, since there is no X-ray evidence, and there is sometimes some recovery of function.

Another group of inner ear disturbances about which we have had very little information regarding the underlying pathology is due to virus diseases. Deafness has been a frequent complication of some contagious diseases, particularly mumps, measles and typhus. Sudden severe inner ear deafness has occurred sometimes in relation to a simple upper respiratory infection with or without a non-suppurative otitis media, or as a part of certain less common syndromes affecting also the ocular apparatus, the salivary glands or the central nervous system and meninges. There has been an almost complete lack of information as to pathogenesis and pathology. The only virus disease in which the histopathology of an inner ear complication has been demonstrated has been measles. We have been able to find only two definitive reports on the pathologic picture produced by the measles virus in the inner ear, and we have recently added one bilateral

case showing inner ear invasion by the measles virus. In this case the finding could be described as an endolymphatic labyrinthitis resulting from direct invasion of the endolymphatic system by the virus by way of the stria vascularis.

In a case reported some years ago by Professor Nager, the evidence indicated that a meningoencephalitis had been present with direct extension from the subarachnoid space to the perilymphatic compartment of the labyrinth. There are also other types of cases in which clinical circumstances suggest that there may have been a direct extension of a virus infection or its toxic effects through the labyrinth windows from the middle ear. The factual information regarding this type of injury to the inner ear is scanty. In this, as in many other diseases of the ear, there is still a paramount need for histopathologic evidence as to what takes place in the inner ear and in the neural apparatus.

One of the diseases which has been most intriguing to the otologist, and also to those of you carrying on research on the function of the cochlea, is that known as Ménière's disease. The characteristics of the auditory disturbances in this condition have been unique. With the accumulation of histopathologic evidence and the development of hearing tests much has been learned about this condition; however, certain features still remain inadequately explained. The basic reason for the hydrops is not settled, and probably will not be finally determined until chemical analysis of the fluids have been further developed. Information pertaining to certain features, such as the recruitment phenomenon, has been obtained through experimental procedures; however, the fluctuating low tone deafness seems to have so far eluded an adequate explanation. Clinical observation has indicated that a Ménière's disease may exist without vertigo and without producing the distortions of the vestibular portion of the labyrinth characteristically found in those ears which have come from patients who had Ménière's disease with attacks of vertigo. Histologic confirmation of this opinion that Ménière's disease may occur without vertigo has been afforded by a case which has recently come through our laboratory. In this particular case there was no history of attacks of vertigo. The character-

istics of the hearing loss, however, were such that a diagnosis of probable Ménière's disease was made. The histologic sections from the affected ear have shown the characteristic hydrops of the cochlear part of the labyrinth, but there was no distortion of the vestibular portion. I am not aware of any other case on record which has afforded definitive information on this particular point.

There are many other problems pertaining to hearing impairment which are far from being adequately explained. One of the greatest problems which has faced the research worker in the field of hearing has been the difficulty in obtaining factual information regarding the pathologic process which has produced the disturbance.

It is obvious that a primary requirement for the understanding of disturbances of function is a complete knowledge of the physiological processes involved in hearing and maintenance of equilibrium. It is, however, evident to the otologist that there are great gaps in information regarding the pathologic states which cause the functional disturbances, and that increased efforts to obtain such inforamtion is an urgent need.

* * *

WEDNESDAY AFTERNOON SESSION.

The International Conference on Audiology convened at 2 o'clock P.M., in the Auditorium of the Washington University School of Medicine, St. Louis, Missouri; Dr. Joseph Erlanger, Washington University School of Medicine, presiding.

NEURAL MECHANISMS IN AUDITION.

ROBERT GALAMBOS,

Walter Reed Army Institute of Research,

Washington, D. C.

My discussion of Listening and Pitch Perception will of

necessity be brief, for in the 30 minutes at my disposal I shall be obliged to paint the picture with a very wide brush. Recently published accounts of these matters are, however, available for those of you who wish more details.[1,3,17,18]

LISTENING.

I am sure all of us here recognize the distinction between "listening" and "not-listening." During this Congress we have heard the phenomenon referred to repeatedly, with such terms as "central deafness," "central factors in deafness" and

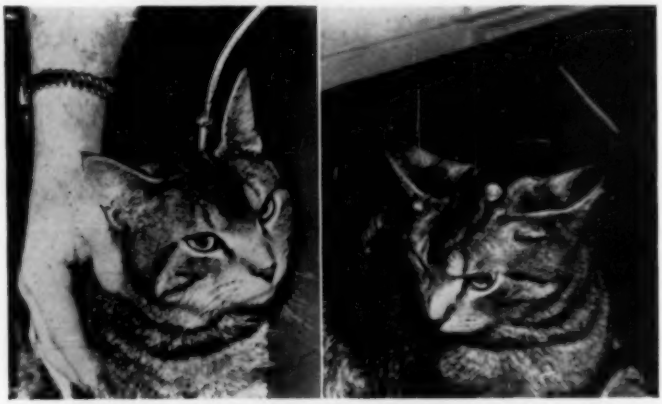


Fig. 1. Cat with eight Implanted Electrodes for E.E.G. Studies.

"hearing but not perceiving" being employed. The main idea behind these concepts is that while the cochlear apparatus within the temporal bone may be entirely normal and fully capable of arousing impulses in the auditory nerve, a central auditory apparatus must also be acting in a special and particular way if sounds are to provoke responses from animals and men. A moment's reflection tells us that the brain must indeed be organized in one way when we listen and in another way when we do not. One of the tasks of the auditory neurophysiologist, it seems to me, is to define as best he can the difference between these two states.

Various attempts to do this have employed cats like that

shown in Fig. 1.[7,9,10] Wire electrodes were implanted in the brain many weeks earlier, and these were led to the outside through the connector seen attached to the skull. In this way the E.E.G. was made available for study whenever desired, and the animal could be examined at any time in the unanesthetized state.

Fig. 2 shows some records from such an animal. The upper line in each section shows the time at which an auditory

Fig. 2. Brain responses to click stimuli from a cat like that shown in Fig. 1. Electrodes 2 through 6 are located 2 mm. apart in the Horsley-Clarke A-18 plane; 6 and 5 are in subcortical white matter, and 4, 3 and 2 are in the caudate nucleus. The size of the brain response obtained to a constant click stimulus seems to depend upon such variables as sleep and attentiveness.

stimulus—in this instance a click of moderate intensity—was presented through a loudspeaker. Notice that the E.E.G. response to this sound is not uniform in size in the cochlear nucleus or the auditory cortex, and that on occasion the stimulus evokes responses from parts of the brain (caudate nucleus) not considered in text books to be part of the auditory system of neurons. The study of many records such as this suggests the conclusion that auditory stimuli of constant strength do

not invariably produce the same effects upon the brain, nor are their effects limited to those brain areas conventionally considered to be auditory in function.

The demonstration that a fixed stimulus may yield a fluctuating brain response leads naturally to the question of whether the response is large when the animal "listens" and less so when it does not. This idea is clearly suggested in

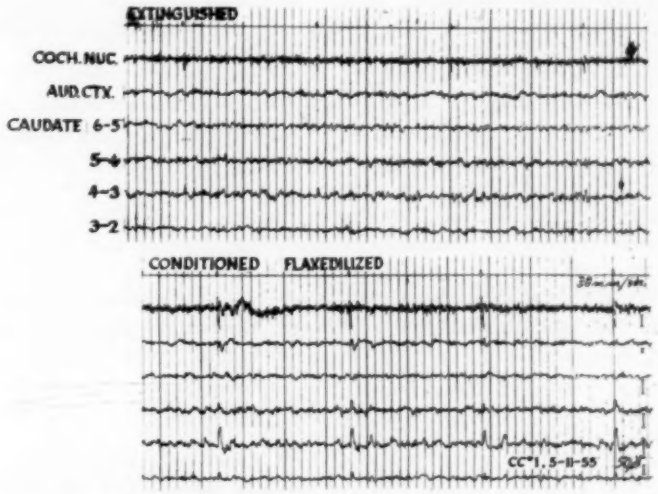


Fig. 3. Effect of a simple conditioning procedure upon the amplitude of response evoked by click. Upper record: small amplitude responses from animal exposed to click day and night for many days; lower record: larger amplitude to same click after it had been paired with shock. To prevent muscle artifacts the animal was treated with a curare-like substance prior to the lower recordings.

Fig. 2, where the cat attends to a visual stimulus; the animal is probably not attending to the click at that time, and the diminished size of the response to clicks may be taken to correspond with this fact.

In the attempt to explore this possibility systematically the recordings of Fig. 3 were made. The records in the lower part of that figure were made after the clicks had been paired

with shocks to the skin. Responses to the clicks are uniformly large in size. It was our intention to condition the cat to expect a shock in the presence of the click, and apparently the clicks did ultimately become a signal for the cat that an unpleasant event was about to occur. The records in the upper part of the figure, on the other hand, had been made at an earlier time when the cat had been in the presence of the same click for many hours, and seemed to be paying no attention whatever to them; the E.E.G. responses to clicks turn out, as expected, to be relatively smaller and considerably less widespread throughout the brain in the habituated as compared to the conditioned state.[7]

The variation in amplitude of the auditory cortical responses in Fig. 3 will come as no surprise, since it is generally thought that the cortex participates in some important way in the attentive function under exploration. The demonstration that much the same phenomenon takes place in the cochlear nucleus, however, demands that the auditory nervous system throughout its entire length be under the control of neural mechanisms that modulate incoming messages, allowing impulses to pass at one time and preventing their passing at others. Unfortunately, we do not completely understand what these controlling mechanisms actually are, but an examination of the known anatomy of the auditory system provides some speculative possibilities.

Fig. 4 shows, on the left, the classical ascending fiber pathways shown in most text books, as well as the newer routes by way of the reticular formation and to the cerebellum that have come to be known over the past 15 years (see[3]). On the right, by contrast, are shown the fiber tracts that descend from the cortex to end up finally in the cochlea itself. This descending system was known in part to Cajal, to Lorente de No and to others of the classical schools of neuroanatomy, but the modern concept of its course and distribution is due largely to the efforts of Dr. Grant Rasmussen. His most notable contributions[12,13] have shown that a bundle of fibers originating in the region of the superior olivary nuclei terminates on or near the internal hair cells. This olivocochlear bundle represents the terminal line in the neural

path that originates at the cortex, descends in close approximation with the classical ascending auditory fibers, and makes synaptic connections in many, if not all of the auditory nuclei. One may speculate that it is through the action of this descending collection of fibers that modulation of the incoming messages occurs, something being "fed" or even "fed back" into the system to provide the control. Such specula-

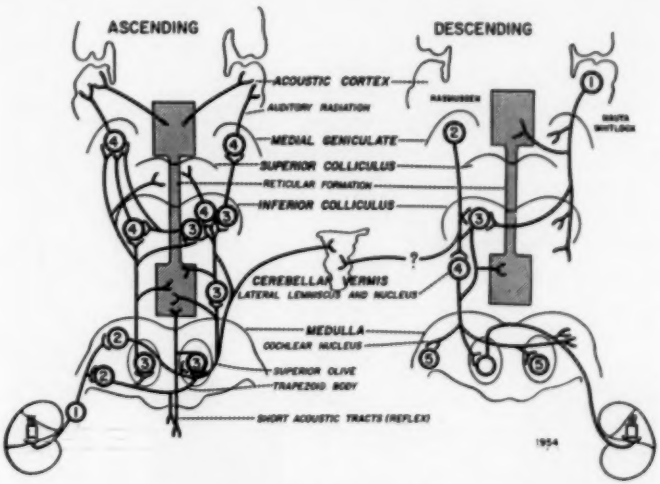


Fig. 4. Diagram of the auditory pathways showing ascending and descending connections.

tions at the present time also involve to varying degrees the limbic system in the forebrain and the reticular formation in the mesencephalon, but these details need not be stressed further. Instead, let us consider some evidence that the olivocochlear bundle of Rasmussen has a definable physiological function in the cat, and by inference in any other animal (including man), with a bundle of Oort in its VIIIth nerve complex.

Fig. 4 summarizes the essential feature of this study,[4] showing that the amplitude of the auditory nerve response to a click stimulus is significantly reduced if a barrage of

impulses is simultaneously arriving at the cochlea via the olivocochlear bundle. This final link in the descending chain of neurons thus has an inhibitory effect upon the auditory system, preventing the arousal of the expected amount of nerve activity to a standard click stimulus. It should be noted that this inhibition was demonstrated by delivering

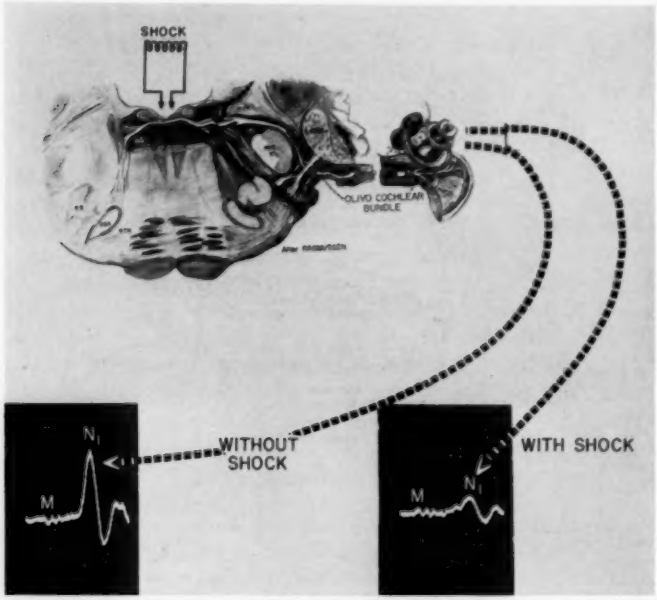


Fig. 5. Anatomical plan of the olivocochlear pathway, and illustration of its function. The cross section shows cells of origin (OSA) and anatomical course of fibers to the cochlear spiral. Shocks applied where the fibers decussate markedly reduce the amplitude of the auditory nerve discharge (N_1) evoked by a click without influencing the hair cell response (M).

electrical shocks to the medulla of anesthetized cats, and that it has not as yet been shown to occur naturally in normal animals; so we cannot be certain in extrapolating these data to the normal case; nevertheless, the final link in the descending system acts as an inhibiting mechanism in these physiological preparations, and from this fact we may, perhaps, be

permitted to infer that the probable role of the entire system in the normal animal is at least in part an inhibitory one.

These first efforts to study the problem of "listening" can be summarized as follows: The attentive animal is characterized by large E.E.G. responses spread throughout its brain, areas being included that ordinarily are not considered to be part of the auditory system. The inattentive animal shows responses in fewer brain locations, and these are small in size. The variation in size occurs throughout the auditory system, being apparent even at the cochlear nucleus, the first synapse in the auditory system. The descending pathway represents a possible neural mechanism for causing the amplitude variations, and its final link, the olivocochlear pathway, is known to function as an inhibitor. Much obviously remains to be done, however, before the problem is finally solved.

PITCH PERCEPTION.

Of all the problems of hearing, that of how we perceive pitch has undoubtedly received the lion's share of attention by experimentalists. Many recent reviews of this topic are available,[1,3,17,18] and so my efforts today need not attempt comprehensiveness. Instead let us consider some old and new studies with microelectrodes that seem to illustrate the major principles thus far elucidated. These microelectrodes, you will recall, allow the action potentials of a single brain cell to be isolated for study. The way the cell responds to the variety of sounds to which the ear is subjected, constitutes the raw data out of which an understanding of the brain is to be fashioned. If we collect data from a sufficiently large number of such cells we can hope to create a complete and satisfactory picture.

Fig. 6 presents some of the earliest records taken from the auditory system with this technique.[5] When the words "VIIIth nerve" were spoken in the vicinity of the cat, a unit in the cochlear nucleus reacted briskly, as is shown there. It has since been repeatedly demonstrated that auditory units in all the auditory nuclei are similarly sensitive to sounds, and when pure tones are employed they respond to a limited and restricted band of them. This fact is demonstrated for

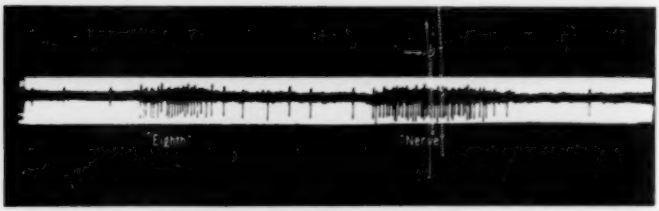


Fig. 6. Response of a single cell in the cochlear nucleus to spoken words. Microelectrode recording from anesthetized cat.

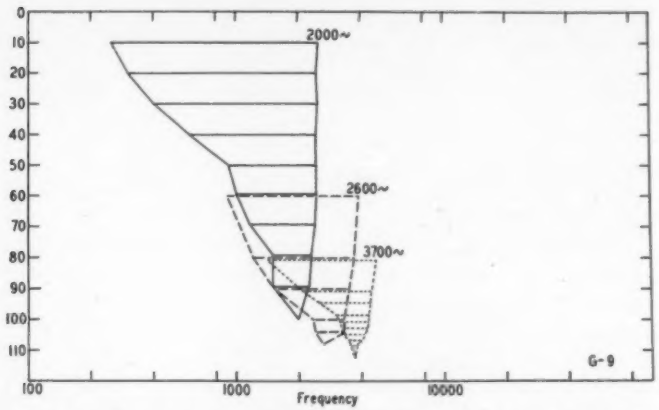


Fig. 7. Response areas for three separate cells in the cochlear nucleus of the anesthetized cat. Ordinate: intensity in db. below reference level for tones given on abscissa. The "best frequency" of each cell is written next to the triangular response area that encloses all tones found adequate for its stimulation.

three units in Fig. 7, where the tones that excite any given unit are seen to fall in a limited part of the sound spectrum. The triangular "response area" thus experimentally defined proves conclusively that auditory units function physiologically in the manner suggested by their anatomical connections, namely, by responding to limited bands of tones. The basic idea of Helmholtz, which was that some unique spatial correlate exists in the brain for each perceived tone is, therefore, essentially confirmed, although the details of how this

comes about is a somewhat more elaborate story than I can give you here (but see [1,3,15,16]).

Fig. 8 shows the experimental demonstration of the place principle in the case of the cochlear nucleus.[14] The path of a microelectrode track is shown there in a saggital section of the nucleus. The electrode entered the dorsal cochlear nucleus

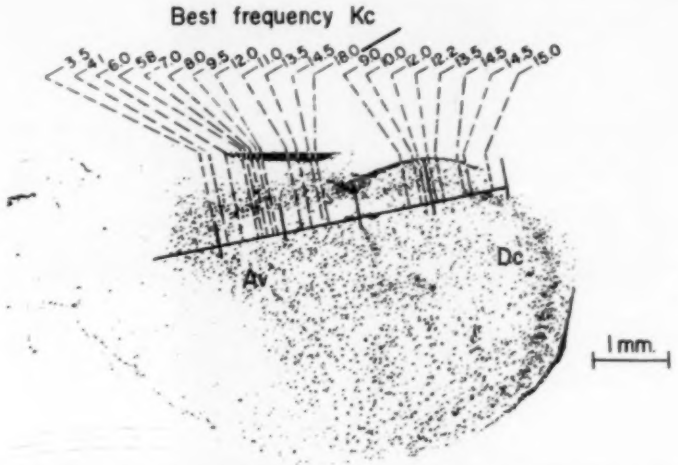


Fig. 8. Plot, on a saggital section of the cochlear nuclei of the cat, of the "best frequencies" encountered during a particular microelectrode puncture. Note orderly decline of frequency in both the dorsal (Dc.), and the anterior ventral (Av.) segments, indicating that projection of the basilar membrane to this nuclear mass is tonotopic.

from behind and proceeded successively through the posterior and anterior ventral cochlear nuclei. The numbers in Fig. 8 represent the tone frequencies found by physiological methods to be best represented at the indicated position. It is clear that an orderly arrangement exists in the various segments of this complex nuclear mass. A similar tonotopic arrangement has been uncovered throughout the auditory system wherever it has been sought experimentally.

We can conclude, therefore, that the evidence is heavy in support of the idea that the place in the brain where a tone

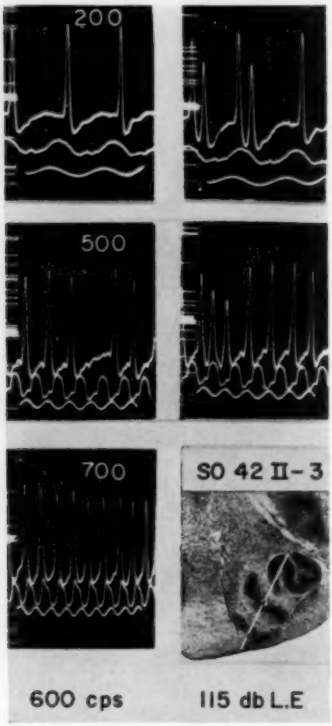


Fig. 9. Responses of a single cell to tones. Anesthetized cat. Electrode location, lower right, accessory superior olivary nucleus. Each segment of the figure shows, at left, the spontaneous activity of the unit, and on the swept traces; the unit response (top line), the cochlear round window response (middle), and the electrical signal delivered to the earphone (bottom. The tone frequency employed is written on the left member of each pair of records to which it pertains. The "best frequency" for this unit was 600 cps.; the stimuli were applied to left ear with recording on the right side.

produces its effects is somehow important for our ability to perceive pitch; but what about that other persistent idea of how tone frequency is handled, namely, that each nerve fiber responds at the frequency of the tone? Fig. 9 shows that, to a remarkable extent, this idea is correct also. The unit pictured there was isolated in the superior olive.[6] It responded to tones up to 1100 cps., and the pictures make it

clear that it often did so by discharging once to each sound wave cycle. The pictures also make it evident, however, that the unit sometimes skipped responding, and that it would on occasion respond twice to a single sound wave cycle. The facts, nevertheless, unequivocally support the idea that tonal frequency can be centrally represented as nerve discharge frequency. We do not as yet know precisely how to evaluate the relative importance of the two principles—place and frequency of nerve discharge—in the mechanism of brain action that results in pitch perception. We can be certain only that

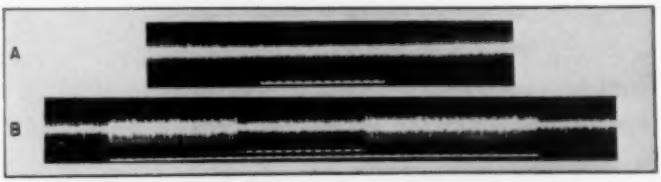


Fig. 10. Suppression of cochlear nucleus cell responses by tones. Anesthetized cat. A.—The spontaneous activity of a unit disappears so long as a tone (white line below record) is on. B.—A unit excited by one tone (long white line) stops completely so long as a second tone (short line) is simultaneously sounding.

the frequency principle must be of limited utility for tones above 1000 or 2000 cps.

Besides the place and frequency principles there are at least two other facts about the auditory brain that almost certainly are relevant to the pitch problem. The first of these is that a low tone activates a larger amount of the auditory brain than does a tone of high frequency. This is true, because the pattern of cochlear motion aroused by low tones spreads from base to apex along the basilar membrane, thus sweeping across the entire neural complement of the cochlea. The pattern evoked by high frequency tones, by contrast, is restricted to the base. The consequences of this fact are not entirely understood, but it is already clear that any partial damage to the VIIIth nerve that involves fibers from the various parts of the basilar membrane in equal

proportions yields a selective loss of hearing for high frequencies.[8,11]

The second ancillary fact is illustrated in Fig. 10. We ordinarily think of the auditory stimulus as creating excitation, or increased nerve activity, when it is presented to the ear. This is certainly the case for many single units in the brain, as can be seen in Figs. 6 and 9. In Fig. 10, on the other hand, what the stimuli do is to suppress the ongoing activity rather than to create more of it (see[2]). This inhibitory effect of tones is a general phenomenon observable at all levels in the auditory system including the cortex.

This demonstration that a particular tone causes certain units to stop firing and others to fire more briskly, probably means that the overall effect of the tone is to create areas of activity interspersed among cellular areas in which activity has been suppressed. Presumably the exact locations and extents of such excited and inhibited areas are of significance as central correlates of the tonal frequency (see[3]), but no clear picture of how this exactly occurs can yet be given.

There are, then, the four factors known to play some role in the central mediation of the experience of pitch. The well-known place and frequency principles clearly are in operation. The "place" involved by a given tone must, however, be conceived as collections of neural loci, in some of which increased activity occurs, whereas decreased activity occurs in others. The configuration of excited and inhibited "places" varies as the tone frequency changes, and this geographical (tonotopic) correlate for pitch is the one most securely established by experimental data. The "places" involved, finally, are more extensive and widespread through the auditory nuclei when the tone is low as opposed to high in frequency. How the central nervous system produces the sensation of a tone out of these bits and scraps of information remains a complete mystery.

REFERENCES.

1. Davis, H.: Biophysics and Physiology of the Inner Ear. *Physiol. Rev.*, 37:1-49, 1957.

2. GALAMBOS, R.: Inhibition of Activity in Single Auditory Nerve Fibers by Acoustic Stimulation. *Jour. Neurophysiol.*, 7:287-304, 1944.

3. GALAMBOS, R.: Neural Mechanisms of Audition. *Physiol. Rev.*, 34:497-528, 1954.

4. GALAMBOS, R.: Suppression of Auditory Nerve Activity by Stimulation of Efferent Fibers to Cochlea. *Jour. Neurophysiol.*, 19:424-437, 1956.

5. GALAMBOS, R., and DAVIS, H.: The Response of Single Auditory Nerve Fibers to Acoustic Stimulation. *Jour. Neurophysiol.*, 6:39-58, 1943.

6. GALAMBOS, R.; SCHWARTZKOPF, J., and RUPERT, A.: Microelectrode Study of Superior Olivary Responses to Sounds. *Fed. Proc.*, 16:43, 1957.

7. GALAMBOS, R.; SHEATZ, G., and VERNIER, V. G.: Electrophysiological Correlates of a Conditioned Response in Cats. *Science*, 123:376-377, 1956.

8. GUILD, S. R.: The Effects on Hearing of Partial Sections of the Cochlear Nerve in Man. *Acta Oto-Laryngol.*, 43:199-207, 1953.

9. HERNANDEZ-PEON, R.; JOUVET, M., and SCHERRER, H.: Auditory Potentials at Cochlear Nucleus During Acoustic Habituation. *Acta Neurol. Latinoamer*, 3:144-156, 1957.

10. HERNANDEZ-PEON, R.; SCHERRER, H., and JOUVET, M.: Modification of Electric Activity in Cochlear Nucleus During "Attention" in Unanesthetized Cats. *Science*, 123:331-332, 1956.

11. NEFF, W. D.: The Effects of Partial Section of the Auditory Nerve. *Jour. Comp. and Physiol. Psychol.*, 40:203-215, 1947.

12. RASMUSSEN, G. L.: The Olivary Peduncle and Other Fiber Projections of the Superior Olivary Complex. *Jour. Comp. Neurol.*, 84:141-220, 1946.

13. RASMUSSEN, G. L.: Further Observations of the Efferent Cochlear Bundle. *Jour. Comp. Neurol.*, 99:61-74, 1953.

14. ROSE, J. E.; GALAMBOS, R., and HUGHES, J. R.: Tonotopic Organization of Frequency Sensitive Units in the Cochlear Nuclei of the Cat. *Anat. Rec.*, 127:358, 1957.

15. TASAKI, I.: Nerve Impulses in Individual Auditory Nerve Fibers of Guinea Pig. *Jour. Neurophysiol.*, 17:97-122, 1954.

16. TASAKI, I., and DAVIS, H.: Electric Responses of Individual Nerve Elements in Cochlear Nucleus to Sound Stimulation (Guinea Pig). *Jour. Neurophysiol.*, 18:151-158, 1955.

17. WEVER, E. G.: "Theory of Hearing," 484 pp., 1949. John Wiley & Sons, Inc., New York.

18. WHITFIELD, I. C.: Electrophysiology of the Central Auditory Pathway. *Brit. Med. Bull.*, 12:105-109, 1956.

PHYSIOLOGICAL DISCUSSION.

ICHIJI TASAKI,

National Institute of Neurological Diseases and Blindness,

Bethesda, Md.

DR. I. TASAKI: Dr. Galambos has just given us a very

interesting survey of the problem of how the nerve fibers and cells in the auditory pathway transmit nerve impulses to the central nervous system. He has shown us clearly that the function of the auditory pathway is not only to transmit messages to the central nervous system but also to assort and classify the messages before they reach the brain, or sometimes, to eliminate some of the messages before they reach the cerebral cortex. Now I should like to discuss the problem before us very briefly from a slightly different angle, namely, in relation to our present day knowledge of more general mechanisms found in the nervous system.

Sometime ago, Dr. Davis and I demonstrated the fact that the auditory nerve fibers arising in the basal turn of the guinea pig cochlea respond to tones of any frequency, high and low, while the fibers originating in the apical turn respond only to low frequency tones (slide). These nerve impulses in the individual auditory nerve fiber obey the all-or-none law. They will not stop until they arrive at (or near) the synapses in their pathway. The behavior of these synapses in the auditory system is probably very similar to that of other excitatory or inhibitory synapses in the nervous system; therefore, physiological studies on the auditory pathway are , in a sense, a sort of experimental study on synaptic transmission in the nervous system. If the pattern of the nerve impulses arising in the cochlea is accurately given, the fate of these impulses will be determined simply by the structure of the nervous system. This part of the problem is not specific to the auditory system.

The problem dealing with initiation of nerve impulses in the cochlea is, on the contrary, very specific to the auditory system, because the structure of the inner ear is very different from that of other mechanoreceptors. Let us take a look at a picture illustrating the familiar structure of the cochlea (slide). When we introduce a glass pipette electrode into various parts in the cochlea, it is found that the electric potential in the space occupied by endolymph is 80-90 mv positive to that of the perilymph (Békésy). It appears to us that the fluid in the space between the basilar membrane and the reticular lamina is 70-80 mv negative to the perilymph.

These dc. potentials can be interpreted as devices for increasing the sensitivity of the nerve fibers to electrical stimuli. Microphonics and other slowly changing potentials are undoubtedly signs of intermediate processes which link the mechanical events with the process of initiation of nerve impulses.

A few days ago Dr. Rasmussen gave us a special strain of guinea pigs, waltzing guinea pigs. He gave us three of them, and we used four; that means we took one more after he left to attend this meeting. These guinea pigs show no sign of microphonic responses. They have no hair cells in the cochlea. The story that the hair cells are responsible for generation of microphonic responses is safe; but, we found a dc. potential of about 80 mv in the endolymphatic space and also a strong negativity in the space between the reticular lamina and the basilar membrane. A few years ago somebody around here made an attempt to attribute the endolymphatic dc. potential in the cochlea to the function of the hair-cells. That was a mistake, my mistake. At present there is no doubt that the positive potential is generated by some oxidative metabolism in the stria vascularis.

In conclusion there is a very intriguing problem in Dr. Galambos' presentation. That is the problem of how the electric responses in the auditory nerve fiber are suppressed by stimulation of the olivocochlear nerve bundle. In general, it is extremely difficult to suppress an all-or-none response in the nerve fiber. All the inhibitory processes that have been demonstrated in other parts of the nerve system take place at the site of initiation of nerve impulses, namely, at the pace-makers.

I am assuming that Dr. Galambos' electrical stimuli were limited to the fibers in the olivocochlear bundle. Under this assumption, the most probable explanation of the phenomenon is to attribute it to secretion of some inhibitory substance which raises the threshold of the nerve endings to mechanical stimuli. A somewhat similar phenomenon has been reported recently by Dr. Leowenstein in the mechanoreceptor in the skin. (*Nature*, 178:1292, 1956).

ANATOMICAL DISCUSSION OF THE PAPER GIVEN BY DR. GALAMBOS.

GRANT L. RASMUSSEN,

National Institute of Neurological Diseases and Blindness,

Bethesda, Md.

I have closely followed the studies of Dr. Galambos during the past several years, and have often wondered about the function significance of this strange system of auditory neurons which course backward in an unorthodox fashion.

It is not necessary to review the general plan of the ascending and descending auditory pathways, since Dr. Galambos has already done this with the aid of his Fig. 4. To be sure his schema of the circuits was necessarily simplified for his presentation.

Our main interest presently concerns the less well-known descending or recurrent conduction chain of auditory neurons which extends from the cerebral auditory cortex to the cochlea itself. It has been possible to reveal this system of fibers by means of axonal and terminal degeneration methods.[1,2,3,4] These fibers are for the most part intermixed with those of the ascending system, which constitutes by far the greater number of fibers. The recurrent fibers hold constant positional relationship and, moreover, are interconnected in an orderly fashion, projecting from definite cell groups of higher levels to particular cells of any one of several auditory nuclei. For example, certain neurons of the inferior colliculus and dorsal nucleus of the lateral lemniscus connect bilaterally with particular cell groups of the dorsal cochlear nucleus, and of the superior olivary complex; thus we see evidence of a certain specificity in the descending system, the meaning of which is not understandable to me at this time.

Having described some of the general anatomical features, I will now present a more detailed description of the pattern of distribution of the descending system, as found at the level of the brain, which holds most interest at this time. I show a

figure of a transverse cut of a cat's medulla oblongata at the level of the olivary complex and olivo-cochlear bundle, which includes also the cochlea. This is similar to Fig. 5, shown by Dr. Galambos, except that here the recurrent fibers coming from the inferior colliculus are displayed.

Here within the confines of the circumscribed area indicated, are scattered fibers which degenerate following a lesion of inferior colliculus and/or dorsal nucleus of the lateral

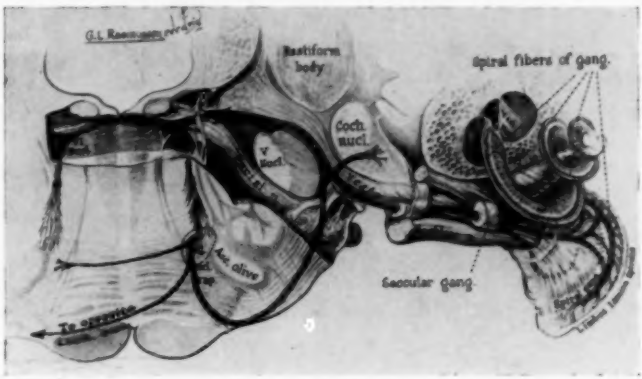


Fig. 1.

lemniscus. Some of these fibers enter and terminate in that part of the olivary complex that has been previously determined as the site of origin of the olivo-cochlear bundle, the fibers of which terminate in the cochlea perhaps about hair cells; however, substantial anatomical proof is still lacking on this point.

While some fibers end in the medial two segments of the olivary complex, a very impressive number of fibers continue on through the trapezoid body to reach and disperse themselves within the dorsal cochlear nucleus of both sides.

This may well be one of the important pathways concerned with suppression of neural activity of the cochlear nucleus from higher levels, as has been mentioned by Dr. Galambos.

This efferent packet of fibers that enters the cochlear nucleus would seem to be identical with that first described by Held,[5] and later by Lorente de Nó,[6] in Golgi preparations. While both recognized its efferent nature, neither were able to determine whence it came.

Finally, I would like to mention that in recent studies I have been able to demonstrate anatomically several other recurrent contributions to the cochlear nucleus that originate from brain regions not usually considered auditory in function.

BIBLIOGRAPHY.

1. Rasmussen, G. L.: The Olivary Peduncle and Other Fiber Projections of the Superior Olivary Complex. *Jour. Comp. Neurol.*, 84:141-220, 1946.

2. Rasmussen, G. L.: Recurrent or "Feed-back" Connections of the Auditory System of the Cat. *Anat. Rec.*, 115:361, 1953.

3. Rasmussen, G. L.: Further Observations of the Efferent Cochlear Bundle. *Jour. Comp. Neurol.*, 99:61-74, 1953.

4. Rasmussen, G. L.: Descending or "Feed-back" Connections of the Auditory System of the Cat. *Amer. Jour. Physiol.*, 183:653, 1955.

5. Held, H.: Die zentrale Gehörleitung. *Arch. f. Anat. u. Entwicklungsgesch.*, 201-248, Leipzig, 1893.

6. Lorente de Nó, R.: Anatomy of the VIIIth Nerve. The Laryngoscope, 43:327-350, 1933.

CLINICAL DISCUSSION OF THE PAPER GIVEN BY DR. GALAMBOS.

J. S. Riesco Mac-Clure,

Neuro-Surgical Institute,

Santiago, Chile.

I am afraid that what I have to say from the clinical point of view is not very satisfactory, because I have been more concerned with vestibular disturbances than with those of the cochlea; however, I will refer to the lesions in the brain stem; lesions due to tumors, encephalitis, hemorrhages and concussions. Between 1950 and 1957 we have seen 76 cases of this kind at the Neurosurgical Institute in Santiago, Chile. The

contrast between the rich vestibular symptomatology and the poor cochlear symptomatology is surprising.

Disorders of the equilibrium, spontaneous nystagmus and positional nystagmus, and marked alterations in the caloric responses were found in almost 100 per cent of the cases. On the other hand, the audiometric examination was always normal, or almost normal, even in those cases in which a bilateral vestibular paralysis was found. Precisely this cochlear and vestibular dissociation, in which the vestibular nerve is paralyzed and the cochlear is normal, is an important sign of a destructive lesion in the vestibular area of the floor of the fourth ventricle.

We have been anxious to see what happens when only the cochlear nucleus at the medulla is involved. We believe that the following case shows something: A 20-year-old man was admitted at the Neurosurgical Institute with the suspicion of a brain tumor. After an exhaustive neurological examination, and after a two-month follow-up, the diagnosis of encephalitis due to virus infection was made. Before becoming ill this young man never complained of hearing loss nor loss of balance.

The audiogram showed a normal left ear and a hearing loss of the ascending type in the right ear with no recruitment, but with diplacusis. At the same time the calorigram showed a total paralysis of the right vestibular branch and paresis of the left vestibular branch. Even more, with water at 18 degrees during a 60 second irrigation a perverted vertical nystagmus was seen.

The patient also had spontaneous nystagmus to the left, a marked loss of balance and involvement of the VIth nerve on both sides. In the absence of recruitment, bilateral cochleo-vestibular dissociation, perverted nystagmus, and neurological involvement of central nature of both VIth nerves, we diagnosed a partially destructive lesion of the floor of the fourth ventricle. Not only a partially destructive lesion of the vestibular area, but also a partially destructive lesion of the right cochlear nucleus, was found.

Apparently these lesions were irreversible, because after six months, although the patient was much better from the neurological point of view, the functional examination of the VIIIth nerve gave exactly the same results.

We believe, then, that the deafness due to a lesion of the cochlear nucleus is very similar to the deafness due to an acoustic tumor. Unfortunately, we have not done the speech

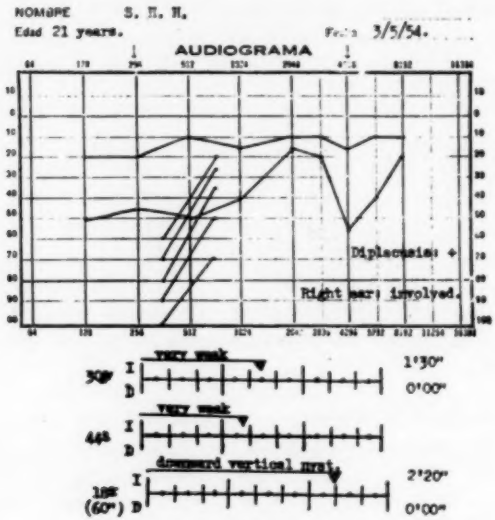


Fig. 1.

tests in our patients because, as I have already pointed out, we have been more concerned with the vestibular system.

Dr. Lindsay has described what he calls "deafness of sudden origin and indefinite etiology." It is possible that some of these cases are due to lesions in the cochlear nuclei.

A 19-year-old woman awakened one morning with strong tinnitus and total deafness in the right ear. She also had some dizziness and loss of balance, but she could continue her school duties. We saw her four months after this had hap-

pened. The otoscopic examination and the vestibular tests were normal.

The audiogram showed a nerve type of hearing loss at right, with absence of recruitment and with a normal differential limen, which does not correspond to a lesion in the end-organ.

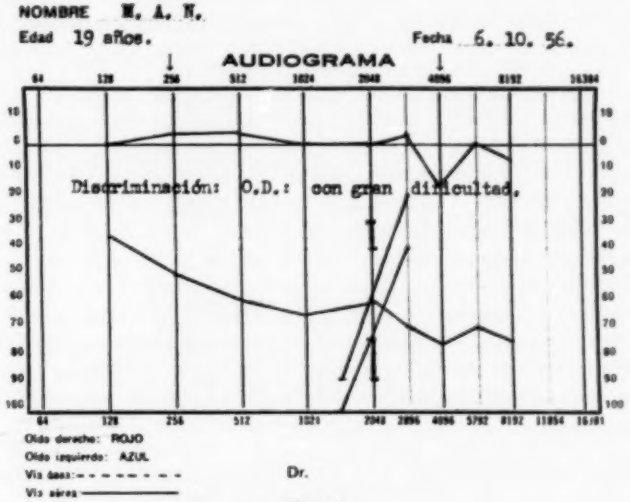


Fig. 2.

The fact that this deafness of sudden origin and indefinite etiology is possible of a complete or partial recovery, as we have seen in a few instances, suggests that the lesion is in the cochlear nucleus and not in the end-organ. Obviously we are not talking about endolymphatic hydrops. When recovery takes place, this happens in the first three weeks; not after. We have seen patients who had a complete unilateral deafness, and within nine to 15 days were totally recovered. We may assume that if the organ of Corti had been damaged no recovery would have been possible.

Patients with stem brain lesions are not numerous, and when one is found, usually its general condition is bad. As

we are asked for a localization diagnosis, we go through all the functional examination of the VIIIth nerve, with particular emphasis on the equilibrium tests, cerebellar and vestibular tests (central and peripheral), which are most important for our purpose; however, we feel now that if the neurosurgeon is not in a hurry, and if the patient is not too bad, we can go through a more complete audiologic examination in order to correlate the clinical findings with the histopathological ones, as we have been doing with the vestibular disorders.

For example, here is a case in which the cochlear study was very poor as compared with the vestibular interpretation. A 47-year-old man had an adenocarcinoma of the thyroid gland with multiple metastasis in the lungs and in the central nervous system. He died before any surgery was performed. The VIIIth nerve examination showed, as usual, a very rich vestibular symptomatology, which led us to the diagnosis of a partially destructive lesion of the right vestibular area on the floor of the fourth ventricle. According to the pathological findings, the diagnosis was correct, and an 8 mm. diameter metastasis had partially destroyed the right vestibular nuclei; but we verified a peculiar hearing loss which we could not interpret.

The audiogram showed that this patient had a slight hearing loss on left ear and moderate deafness on the right, with a Weber lateralized to the right. The middle ears were normal. The report of the pathologist showed that a small metastasis involved the right olive in the medulla.

Another case, a 42-year-old man, had a clear symptomatology of a brain tumor: headaches, vomiting and very marked papillo-edema, but the neurologists were doubtful whether the lesion, the tumor, was in the anterior or posterior cerebral fossae.

If the neurological symptomatology was puzzling, the results of the VIIIth nerve examination were even more difficult to understand.

He had no hearing at left, but the vestibular branch, ac-

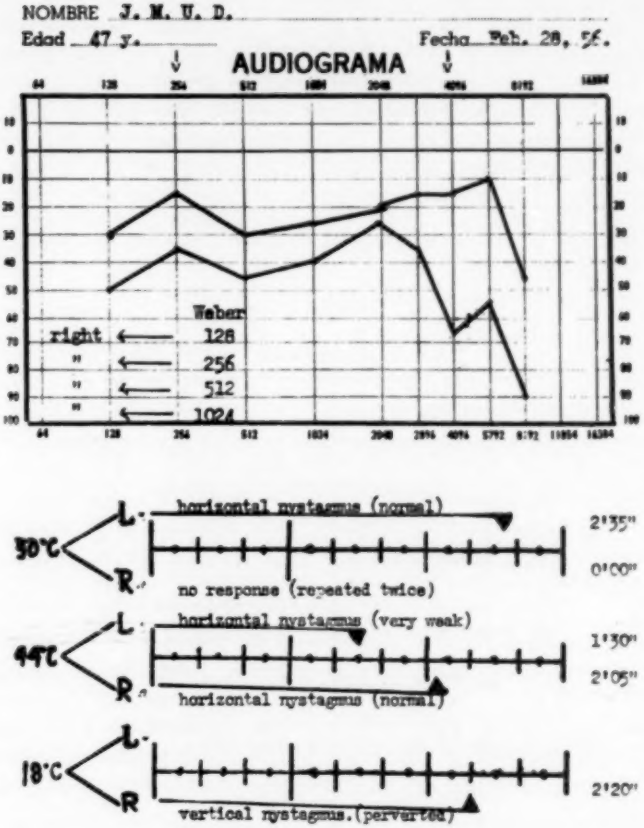


Fig. 3.

cording to the caloric response was normal. On the right ear he had a moderate hearing loss, but no vestibular response. Besides this inverted dissociation, he also had: *a.* spontaneous nystagmus to the left; *b.* positional nystagmus; *c.* complete loss of balance—could not stand upon his feet; *d.* bilateral hypotony; and, *e.* ataxia in the finger-nose tests and adiadokokinesia. These cerebellar symptoms were bilateral.

From time to time we get cases such as this, in which the otoneurological diagnosis is almost impossible, because there is a cochlear paralysis with normal vestibular in one ear, and a cochlear paresis with a vestibular paralysis in the opposite ear. As though this were not enough, the patient had a definite central vestibular symptomatology and bilateral cerebellar symptoms.

You may ask: what did the electroencephalogram show? It showed abnormal activity in the right cerebral hemisphere.

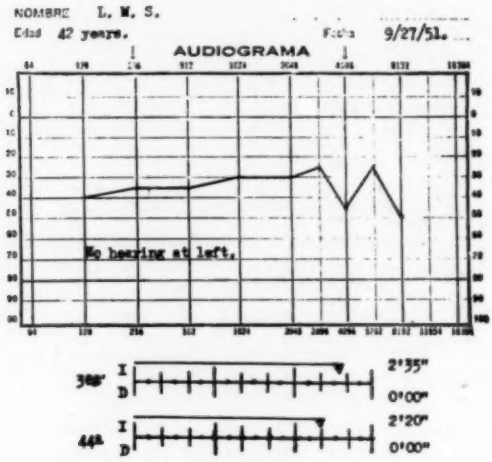


Fig. 4.

At the necropsy a large astrocytoma in the right cerebral hemisphere was found. It was a large, deeply located tumor which strongly compressed the brain stem at the incissura tentori. This was undoubtedly the cause of the central vestibular symptomatology. The pathologist found that the tumor had invaded the right medial geniculate body and considered this lesion responsible for the left total deafness and right partial deafness.

The contrast between the rich vestibular symptomatology

and the poor cochlear symptomatology in cases of brain stem lesions is surprising. I should say rather that it is surprising the contrast between the attention we have given the vestibular disorders and the very reduced interest we have shown in the cochlear disorders.

If any conclusion can be drawn out of these clinical observations, it is that, until the present moment, very few efforts have been made to learn what actually happens to the auditory function when there is a lesion of the brain stem.

REFERENCES.

LINDSAY, J. R.: Inner Ear Deafness of Sudden Onset. THE LARYNGOSCOPE, 60:238, March, 1950.

FUNCTIONS OF THE AUDITORY CORTEX.

WILLIAM D. NEFF,

University of Chicago,*

Illinois.

Because of the lack of appropriate clinical material, most of our knowledge of the functions of the auditory projection areas of the cerebral cortex must come from experiments done on animals other than man. In the last fifteen years, considerable progress has been made in animal investigations. Several factors have contributed to this progress:

1. The use of electrophysiological recording and stimulating techniques to define more exactly the auditory pathways and centers in the central nervous system; *2.* the use of new techniques of experimental surgery; *3.* the development of improved methods of testing auditory discrimination; *4.* the application of standard neuroanatomical procedures to discover new details of cortical and thalamo-cortical organization.

*Research on hearing, which is done in The Laboratory of Physiological Psychology, is supported by the Office of Naval Research and by a grant from the Sonics Research Foundation.

In the following paragraphs some of the results of recent studies which have investigated the role of the auditory cortex in sound discriminations, are briefly summarized. Particular attention will be given to experiments done in our laboratory with only occasional reference to related studies done by other investigators. Emphasis will be placed upon experiments, in which behavioral methods were used to test auditory discriminations before and after partial or total ablation of auditory cortex. Since the auditory nervous system of the cat has been more thoroughly explored than that of any other animal, I shall confine most of my discussion to studies in which the cat was used as experimental subject. Brief

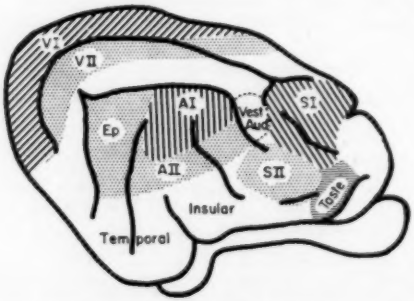


Fig. 1. Cerebral cortex of the cat, showing auditory areas I, II, and Ep, the insular-temporal region, and projection areas of other sensory systems.

mention will be made of parallel studies which have been done on the monkey.

The auditory cortex, as it has been defined for recent experiments, is shown in Fig. 1. It consists of the areas labelled AI, AII, and Ep. This map is based upon results from electrophysiological, cytoarchitectural, and retrograde degeneration studies.[1,2,3,4]

In 1943, Kryter and Ades[5] reported that the absolute thresholds of the cat, as tested in a conditioned avoidance situation, were unchanged by bilateral ablation of a region which included all of AI, AII and Ep. They made tests only in the frequency range from 125 c.p.s. through 8000 c.p.s.,

but there is no later evidence to suggest that losses would have occurred for higher frequencies which the cat hears.* A loss of the learned habit occurred as a rule after the bilateral cortical ablations, but relearning was at least as rapid as initial learning.

In similar studies, Raab and Ades[7] and Rosenzweig[8] have shown that no loss in capacity to discriminate small changes in intensity occurs after bilateral ablation of AI, AII, and Ep. Again the learned habit is affected, but post-operative relearning is rapid.

Several studies have shown that the auditory cortex of mammals (at least of the cat, dog and monkey) has a tonotopic organization, *i.e.*, as measured by electrophysiological recording from gross electrodes a given point on the cortex has a lower response threshold for a particular frequency, or narrow band of frequencies, than for other frequencies in the audible range.[9,4,10] Because of this tonotopic organization, it has often been suggested that the auditory cortex may be particularly important for discrimination of changes in frequency.

Experiments done by Meyer and Woolsey,[12] and by Butler, Diamond, and Neff,[13] have shown that this is not true. Fine discriminations of frequency can be made after complete bilateral removal of AI, AII, and Ep.

For a number of reasons, a particularly important one being a consideration of experimental evidence on functions of visual cortex, it might be inferred that the auditory cortex plays an important part in pattern discrimination. This inference is true, at least for discrimination of temporal patterns of tones. Diamond and Neff[14] have shown that after complete bilateral ablation of AI, AII, and Ep, cats cannot discriminate two tonal patterns, one consisting of three tonal pulses arranged in the sequence, low-high-low, and the other of the same tones presented in the sequence, high-low-high. If a region corresponding approximately to AI is ablated bilaterally, the animal can make this pattern discrimination

*It has been shown that cats can learn to respond to tones at least as high as 60,000 c.p.s.[6]

and, in fact, shows no post-operative loss of the learned habit. If all but a small remnant of auditory cortex is ablated bilaterally, there is amnesia for the learned habit, but relearning occurs. Jerison and Neff[15] have reported similar results for the monkey.

Another kind of discrimination, in which a deficit has been shown to occur after ablation of auditory cortex, is localization of sound in space. Neff, Fisher, Diamond, and Yela,[16] and Neff, Arnott, and Fisher,[17] have shown that cats are deficient in ability to localize sound in space after bilateral ablation of all or even of part of the auditory areas I, II, and Ep. After complete ablation of these areas, an animal will localize accurately an angle of about 40 degrees, whereas the normal animal, in the same situation, will localize a sound (buzzer) when the angle between separate positions in which it is presented successively is as small as 5 degrees.

From the post-operative study of the brains of animals used in some of the studies described above, and from studies done by Rose and Woolsey,[2] a fairly clear picture of the gross thalamo-cortical relationships has been obtained. After ablation of AI there is complete, or nearly complete, degeneration of the anterior half (approximately) of the principal part of the medial geniculate body. After ablation of AI, AII, and Ep the degeneration extends posteriorly to include the anterior two-thirds of the geniculate. Degeneration in the posterior part of the geniculate is found only when the ablation is extended into the insular and temporal cortex ventral to AII and Ep. In an unpublished anatomical study, Diamond, Chow, and Neff have shown that some degeneration of the posterior third of the medial geniculate occurs after experimental ablations confined to the insular-temporal region. From a careful analysis of the retrograde degeneration found in the medial geniculate after cortical ablation, it becomes clear that there is more than one kind of projection from the medial geniculate to the cortex. AI receives what Rose and Woolsey have termed "essential" projection, while AII, part of Ep, and the insular-temporal region receive "sustaining" projection; however, this two-category division is not sufficient, since it appears that the kind of projection

to AII, Ep, and to the insular-temporal region may not be the same.

Attention having been called to the insular-temporal cortex because of its anatomical relationship to the medial geniculate body, experiments have been started to determine the role it may play in auditory discrimination. Results, which are not yet complete, indicate that frequency discriminations can be made after complete bilateral ablation of AI, AII, Ep, and the insular-temporal region (in some cases the ablations were also extended anteriorly in the ectosylvian gyrus to include somatic area II). A very surprising finding has been that a severe, and perhaps complete, loss of pattern discrimination occurs after bilateral ablation of the insular-temporal cortex alone, areas AI, AII, and Ep being left intact.[18] This finding suggests that the insular-temporal region serves a function considerably more important than that of a receiving area for afferent impulses. Its integrity may be essential for the normal functioning of the other auditory cortical areas, or it may have to do with the transmission and utilization of auditory information which is received and elaborated in areas I, II, and Ep. A region homologous in terms of its relations anatomically to the auditory system and its role in auditory discrimination remains to be discovered in other mammals. The identification of such a region in the monkey or other primate will be of particular interest.

The cursory review which I have given of experimental studies of the auditory cortex is sufficient, I hope, to indicate that the so-called auditory cortex is not a unitary region, whether considered from the standpoint of its structural relationships or of its function. We have a reasonable amount of knowledge about the afferent projections of the auditory system to the subdivisions of this region; we know very little, however, about its connections with other systems, *e.g.*, with the unspecific afferent systems, with the limbic system, and with the recurrent or efferent auditory system to which our attention has been called by the experiments of Grant Rasmussen.[18,19,20]

In my opinion, we have made considerable progress in

understanding something of the role of the total auditory region in sensory discrimination, but our analysis of the functions of the sub-areas is only beginning. This must be kept in mind when considering the relationship between the results of experimental studies and clinical studies of man. In the latter, complete bilateral destruction of auditory projection areas probably never occurs. Lesions produced by accident, disease or surgery are not likely to be confined to any one subdivision, nor even to auditory cortex, and the exact locus of the lesion can seldom be determined by post-mortem examination; nevertheless, if experimental studies of animals are extended in directions such as I have suggested, and, if use is made in the clinic of tests which measure functions similar to those which can be tested in lower animals, a gradual and profitable integration of findings from research in the experimental laboratory and in the clinic should be possible.

BIBLIOGRAPHY.

1. Woolsey, C. N., and Walzl, E. M.: Topical Projection of Nerve Fibers from Local Regions of the Cochlea to the Cerebral Cortex of the Cat. *Johns Hopk. Hosp. Bull.*, 71:315-344, 1942.

2. Rose, J. E., and Woolsey, C. N.: The Relations of Thalamic Connections, Cellular Structure and Evokable Electrical Activity in the Auditory Region of the Cat. *Jour. Comp. Neurol.*, 91:441-466, 1949.

3. Bremer, F.: Analyse Oscillographique des Réponses Sensorielles des écorces Cérébrale et Cérébelleuse. *Rev. Neurol.*, 87:65-92, 1952.

4. Hind, J. E.: An Electrophysiological Determination of Tonotopic Organization in Auditory Cortex of Cat. *Jour. Neurophysiol.*, 16:475-489, 1953.

5. Kryter, K. D., and Ades, H. W.: Studies on the Function of the Higher Acoustic Nervous Centers in the Cat. *Amer. Jour. Psychol.*, 56:501-536, 1943.

6. Neff, W. D., and Hind, J. E.: Auditory Thresholds of the Cat. *Jour. Acoust. Soc. Amer.*, 27:480-483, 1955.

7. Raab, D. H., and Ades, H. W.: Cortical and Mid-Brain Mediation of a Conditioned Discrimination of Acoustic Intensities. *Amer. Jour. Psychol.*, 59:59-83, 1946.

8. Rosenzweig, M.: Discrimination of Auditory Intensities in the Cat. *Amer. Jour. Psychol.*, 59:127-136, 1946.

9. Tunturi, A. R.: Audio Frequency Localization in the Acoustic Cortex of the Dog. *Amer. Jour. Physiol.*, 141:397-403, 1944.

10. Kennedy, T.; Kiang, N., and Sutton, S.: Auditory Projection Areas of Cortex in Monkey (Macaca mulatta). *Amer. Jour. Physiol.*, 183:634, 1955.

11. MEYER, D. R., and WOOLSEY, C. N.: Effects of Localized Cortical Destruction upon Auditory Discriminative Conditioning in the Cat. *Jour. Neurophysiol.*, 15:149-162, 1952.

12. BUTLER, R. A.; DIAMOND, I. T., and NEFF, W. D.: Role of Auditory Cortex in Discrimination of Changes in Frequency. *Jour. Neurophysiol.*, 20:108-120, 1957.

13. DIAMOND, I. T., and NEFF, W. D.: Ablation of Temporal Cortex and Discrimination of Auditory Patterns. *Jour. Neurophysiol.*, 20:300-315, 1957.

14. JERISON, H. J., and NEFF, W. D.: Effect of Cortical Ablation in the Monkey on Discrimination of Auditory Patterns. *Fed. Proc.*, 12:237, 1953.

15. NEFF, W. D.; FISHER, J. F.; DIAMOND, I. T., and YELA, M.: Role of Auditory Cortex in Discrimination Requiring Localization of Sound in Space. *Jour. Neurophysiol.*, 19:500-512, 1956.

16. NEFF, W. D.; ARNOTT, G. P., and FISHER, J. F.: Function of the Auditory Cortex: Localization of Sound in Space. *Amer. Jour. Physiol.*, 163:739, 1950.

17. GOLDBERG, J. M.; DIAMOND, I. T., and NEFF, W. D.: Auditory Discrimination After Ablation of Temporal and Insular Cortex in Cat. *Fed. Proc.*, 47:204, 1957.

18. RASMUSSEN, G. L.: Olivary Peduncle and Other Fiber Projections of Superior Olivary Complex. *Jour. Comp. Neurol.*, 84:141-220, 1946.

19. RASMUSSEN, G. L.: Further Observations of the Efferent Cochlear Bundle. *Jour. Comp. Neurol.*, 99:61-74, 1953.

20. RASMUSSEN, G. L.: Recurrent or "Feed-back" Connections of the Auditory System of the Cat. *Amer. Jour. Physiol.*, 183:653, 1955.

DISCUSSION OF FUNCTION OF AUDITORY CORTEX.†

ARCHIE R. TUNTURI,

University of Oregon,

Portland, Ore.

INTRODUCTION.

A decade ago you would not have believed what has been presented today concerning the auditory system, and the auditory cortex in particular. This modern research into the auditory system was initiated by the Office of Naval Research, to whom we owe a special debt for the contributions which

†This investigation was performed under Contract Nonr-1779 (01) (NR 140-103) between the University of Oregon Medical School and the Office of Naval Research. It was supported also by a research grant (B-45) from the National Institute of Neurological Diseases and Blindness. Reproduction in whole or in part is permitted for any purpose of the United States Government.

you have heard. Dewey Neff has presented an outstanding review of the research on the auditory cortex which presents many problems, and their solutions are slow in being realized. Bit by bit the knowledge of the anatomy and physiology of the auditory cortex is accumulating, so that we have now a more advanced understanding of this region of the brain; however a serious need is a discipline which will permit a break through into the function of the auditory cortex, and more generally into the function of the brain as a whole. We would like to hope that this discipline is attainable within the next decade. The need for knowledge of how the brain functions is important, not only to the understanding of the relation of the auditory cortex to speech perception and hearing but also to an understanding of the brain in neurological and mental diseases. I believe that you will agree with me after listening to these excellent papers today, that the audiometer after all tests but a tiny portion of our hearing.

We are indebted for our knowledge of the minute anatomy of the nerve cells in the auditory cortex to Cajal[2] and Lorente de No,[3] who have applied the Golgi silver techniques to unravel the temporal region. The nerve cells in the auditory cortex are so densely packed that the detailed course of the individual nerve connections cell by cell is still practically unattainable. The nerve fibers from the medial geniculate body to the cortex have been explored grossly by retrograde degeneration techniques. Some facts are known of the nerve fibers arising from the other parts of the brain and terminating in the auditory cortex. Brodman's[1] parceling of the cerebral cortex into areas has proved of doubtful value to the modern neurophysiologist.

Physiological studies have led to more precise location of the projections from the geniculate body upon the cortex, and in particular the detail within these connections.[6,7,8,9,10,11] We have data on the time it requires nerve impulses to travel from ear to cortex, the distribution of responses over the surface, and the cell layers where the responses arise. In ablation experiments performed on various animals we are faced with the surprising fact that surgical lesions of the auditory cortex produce little effect on conditioned reflexes.[12] Removal

of the auditory cortex causes no change in the threshold of hearing, multiple conditioned reflexes are not affected, but Dewey[4,5] has presented evidence for disturbances in conditioned reflexes to temporal aspects of stimuli. The search for psychological correlates of hearing has been in vain. Evidence for space localization of sounds, pitch discrimination or difference limens of intensity or pitch has not been found. Perhaps there is some evidence for a loudness function.

Thus far we have failed to implicate the auditory cortex with a specific function. Let us consider more closely the meaning of function. We all like to apply the term function in terms of our own behavior, or something that we can grasp simply. Suppose in this case we consider the function in terms of the central nervous system. With this in mind, let us examine certain stable properties of the auditory cortex that do not change under various conditions. One such property is the frequency localization in which a response to a particular frequency of a sound always occurs in the same location.[13] The spacing between the foci of response is constant, giving us what might be called a frequency map of the auditory cortex. The time of arrival as well as the time course of the responses remain constant. The masking of the cortical responses by means of steady tones or noise is extremely stable and accurately measureable.[14] The masking data from the cortex are not unlike the results obtained in human subjects. The threshold curve at the auditory cortex, even under anesthesia, is identical to the normal thresholds of human hearing. Other constant properties are the refractory period, the absence of contralateral masking, the excitation of a strip of cortex at one intensity for one ear but not the other, and the refractory period between responses due to sounds delivered to the opposite ears.

How the auditory cortex functions must be based upon the structural and physiological properties that remain stable. The nervous system does not hear what is occurring at the ear, but only the electrical activity in the nerve cells of the pathways and the auditory cortex. It is a necessary premise that our acoustical environment must be represented in a reasonable form in the central nervous system, a conclusion

not unlike that of Locke, Berkley and Hume, that our external world is limited to our own central nervous system. Our interests in stable properties is that they alone can form the representations of the external environment. The representations form a copy of the acoustical world, and it is with this copy that the nervous system must concern itself. If the copy is not stable nothing would be known of the external world. Since we are dealing with copies or representations, albeit in coded form, we are most certainly dealing with communication.

In our study of these copies of our external environment, a region has been found in which after much simplification there exists a memory for over one-and-a-half billion different sounds,[15] a memory lasting as much as 200 times the duration of sound itself. This portion of the auditory cortex is no longer than three-quarters inch and no wider than one-half inch. Yet to this region of the brain no psychological correlate, nor function in terms of behavior, has been assignable.

What then is the function of this area to the nervous system itself? What we would like to do is study the effects of the area on messages or information. By studying the relation of acoustical signals to the representations it has become possible to establish a mathematical formulation of the communication aspects of the auditory cortex. This allows up to compute the amount of information received at the auditory cortex in various sounds; therefore, might not communication theory be the discipline we need to understand the function of the brain? After all, the brain uses up more information than any other substance. None of us would believe that the mind consists of mitochondria, proteins, sugars, acetylcholine, choline esterases or other enzymes. These materials are like the junction of two metals to a transistor, or what oil is to a relay, but have no bearing on the information handled by such devices. Needless to say the brain has been compared to computers, but in many ways it has properties different from these mechanisms. I, therefore, ask you to watch the developments in information theory toward a solution of the problem of the brain.

Study of the anatomy and physiology of the auditory cortex

has uncovered a multitude of new facts during the last decade concerning its structure and performance. Conditioned reflex studies have ruled out many possible functions of the area but have suggested other ones. What is needed is a new discipline or scientific method which will give us the answer to the function of the brain. Such a method we feel is very likely available in communication theory.

BIBLIOGRAPHY.

1. Brodman, K.: "Vergleichende Loakalisationslehre der Grosshirnrinde." Verlag Von Johann Am- Brosius Barth, Leipzig, 1925.

2. Cajal, S. Ramon: "Histologie du Systeme Nerveux de l'Homme und des Vertebrates," 2 vols. A Maloine, Paris, 1911.

3. Lorente de No, R.: "Physiology of the Nervous System." J. F. Fulton. Oxford Medical Publication, 274 pp., New York, 1943.

4. Neff, W. D., and Diamond, Irving T.: Role of Auditory Cortex in Discrimination of Tonal Patterns. *Federation Proceedings*, 12, 1953.

5. Neff, W. D., and Jerison, H. J.: Effect of Cortical Ablation in the Monkey on Discrimination of Auditory Pattern. *Federation Proceedings*, 12, 1953.

6. Tunturi, Archie R.: Audio Frequency Localization in the Acoustic Cortex of the Dog. *Amer. Jour. Physiol.*, 141:397-403, 1944.

7. Tunturi, Archie R.: Further Afferent Connections of the Acoustic Cortex of the Dog. *Amer. Jour. Physiol.*, 144:389-394, 1945.

8. Tunturi, Archie R.: A Study on the Pathway from the Medial Geniculate Body to the Acoustic Cortex in the Dog. *Amer. Jour. Physiol.*, 147:311-319, 1946.

9. Tunturi, Archie R.: Physiological Determination of the Boundary of the Acoustic Area in the Cerebral Cortex of the Dog. *Amer. Jour. Physiol.*, 160:395-401, 1950.

10. Tunturi, Archie R.: Physiological Determination of the Arrangement of the Afferent Connections to the Middle Ectosylvian Auditory Area in the Dog. *Amer. Jour. Physiol.*, 162:489-502, 1950.

11. Tunturi, Archie R.: A Difference in the Representation of Auditory Signals for the Left and Right Ears in the Iso-Frequency Contours of the Right Middle Ectosylvian Auditory Cortex of the Dog. *Amer. Jour. Physiol.*, 168:712-727, 1952.

12. Tunturi, Archie R.: Effects of Lesions of the Auditory and Adjacent Cortex on Conditioned Reflexes. *Amer. Jour. Physiol.*, 181:225-229, 1955.

13. Tunturi, Archie R.: Analysis of Cortical Auditory Responses with the Probability Pulse. *Amer. Jour. Physiol.*, 181:630-638, 1955.

14. Tunturi, Archie R.: Masking of Cortical Responses in Middle Ectosylvian Auditory Area of the Dog. *Amer. Jour. Physiol.*, 184:321-328, 1956.

15. Tunturi, Archie R., and Dudman, John A.: Model of the Storage Space in the MES Auditory Cortex. In publication.

FUNCTION OF THE AUDITORY CORTEX. CLINICAL DISCUSSION (NEUROSURGERY).

ROBERT B. KING,*

State University of New York,

Syracuse, N. Y.

In discussing the functions of the auditory cortex from a clinical standpoint, a subject so ably presented by Dr. Neff in "Parameters of controlled physiological investigation," I must confess that little definitive clinical work has been undertaken. There are, however, many areas of mutual concern to physiologist, anatomist, physicist and physician; indeed, even surgeon. As Dr. Neff has said, there is often a 5 to 10 year gap between concepts of function demonstrated in laboratory environs and their initial adaptation for clinical use. A major challenge to all of us must be to absorb this time deficit by earlier synthesis of laboratory analysis, and thereby to gain understanding of non-clinical investigative findings which are of clinical consequence. Clinical perplexities, however, examined in the arena of the investigator, will yield fruitful avenues for investigation of fundamental mechanisms in anatomy, physiology and psychiatry. The interchange of experience should be mutual.

Recent and current studies of auditory cortex with parameters of neurophysiology and psychology have been discussed today. Although clinical efforts to understand the functioning of auditory cortex in more than broad and general terms have had a late beginning, yet three avenues of clinical study are of current interest.

Detailed examination of discrimination functions have been reported by Bocca, and were discussed in detail yesterday.

In a similar vein, discriminative functions have also been studied in six patients with infantile hemiplegia before and after hemispherectomy:

1. Pure tone audiograms were normal. A list of spondees

*Formerly at Washington University, St. Louis, Mo.

was presented to each ear of these patients, and threshold intensity established for the correct identification of 50 per cent of these bisyllabic words. At 70 db above this threshold, then, two series of monosyllabic test words were delivered separately to each ear. A distinct discrepancy between the left and right was clearly evident in such testing of discriminatory function. Forty-five per cent of test words delivered to the ear contralateral to the abnormal hemisphere were incorrectly identified, while 20 per cent of the words so delivered to the ear contralateral to the more normal hemisphere were not repeated correctly. This 20 per cent may indicate a wide variation of normal related to the relatively normal hemisphere.

Although the post op. E.E.G. in this hemisphere became more normal, and there was improvement in seizures and behavior disorders in these patients, there was no improvement in their postoperative discrimination scores. This persisting deficit suggests that nociferous cortex of the abnormal hemisphere was not contributing materially to the deficit inferred for the more normal hemisphere. It may be that this degree of abnormality represents the maximum potential for discrimination of a single hemisphere. Further experience with such testing techniques as these may well yield useful methods for better clinical understanding of focal cortical lesions of auditory and related association cortex.

A second avenue of clinical investigation has been that which Penfield and his co-workers[2] have explored. The stimulation of abnormal cortex with patients under local anesthesia may initiate an auditory experience. Tinnitis may be reported with stimulation of the superior temporal gyrus. From neighboring regions, complex, and at times prolonged auditory sequences, songs or even symphonic orchestrations, may be reported by the patient. In this circumstance, however, a combination of memory and recall of auditory events add to the complexity of the analysis. Although the stimulus in such a circumstance as this is quite an artificial one, the experience described by patients may be realistic in many details.

There has been no extensive study of the electrophysiologic

potentials evoked by such stimuli in man. A third avenue of investigation then is being explored. Single and multiple evoked responses of human cortex are being studied and will be reported at a later date.[3] Such evoked potentials indicate that there may be a number of regional variables in human cortex which do not appear in studies of other cortex.

Fig. 1 illustrates evoked potentials from human frontal association cortex and from the superior temporal gyrus. A calomel cell recording electrode on the pia was recorded

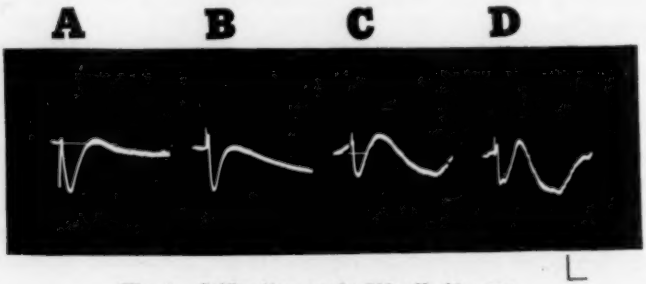


Fig. 1. Calibration angle 500; V. 20 msec.

against a remote electrode. Stimuli for "A" were delivered at the surface 3 mm. from the recording electrode. The potential indicates a pure dendritic response. Stimuli for "B" were delivered at the subcortical white matter of the same gyrus just below the surface recording electrode (radiation response). In "C" the stimulus was applied to subcortical white matter of the adjacent gyrus activating "U" fibers. The surface of the superior temporal gyrus was recorded in "D," while stimulating adjacent white matter (radiation response). The response in "B" and "D" are then analogous. The increased complexity of the potential form in the later instance is apparent. The prolonged late negative deflection in "D" has not been seen in records taken from a limited number of other cortical regions.

Further comparison of the evoked cortical response in frontal and temporal association areas can be illustrated in

the next two slides. The first (see Fig. 2) illustrates evoked radiation responses from frontal association cortex (as in Fig. 1B) with repetitive stimulation (A—6/sec.; B—10/sec.; C—20/sec.; D—50/sec., and E—50/sec.) using DC amplifiers. Note the negative shift in steady potential with increasing frequency of stimuli, and the persistence of this shift after terminating the stimulus.

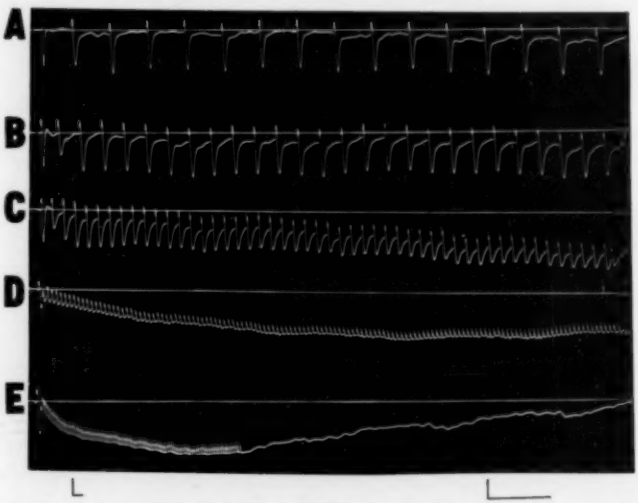


Fig. 2. Right angle at right calibration for E-500; V, and 1 sec. Right angle at left is calibration for A-D 50; V, and 20 msec.

Similar repetitive stimulation of subcortical white matter of the superior temporal gyrus (see Fig. 3) shows further changes not seen in recordings from frontal association cortex. The increasing complexity of this response is apparent as the frequency of stimulation increases (B—6/sec.; C—10/sec.; D—20/sec.; E—50/sec.; F—100/sec.) In "B" and "C" a negative shift similar to that shown in Fig. 2 for frontal association cortex is apparent, but in "D," "E," and "F," an early positive shift of the base is the dominant change returning to the original base line. The negative shift persists after the period of stimulation at lower frequencies.

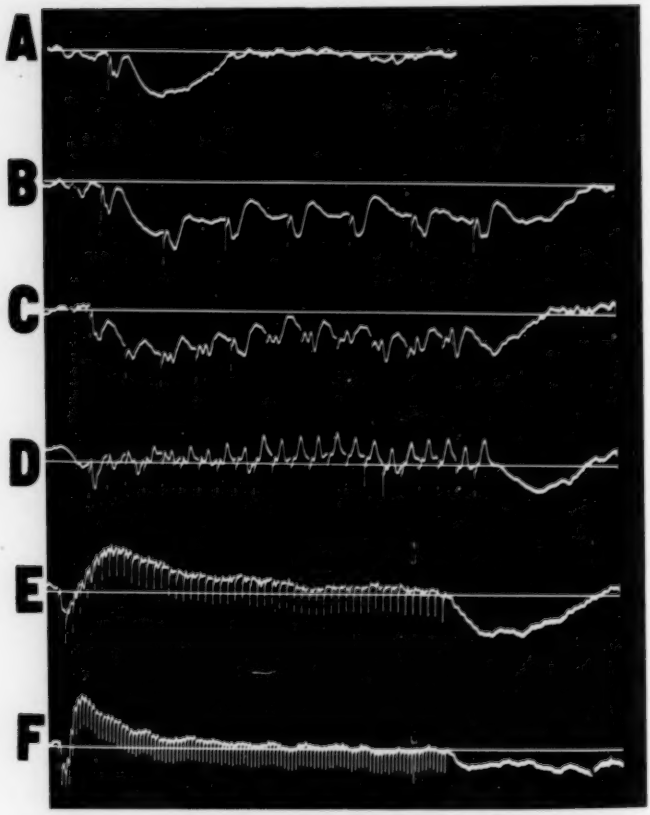


Fig. 3. Vertical celibration 500; V Horizontal calibration 20 msec.

A detailed analysis of these evoked cortical potentials is not warranted at present. Many more records must be acquired before a synthesis, based on such an analysis, can have meaning in terms of the functioning of neural components of frontal and temporal association cortex. This third avenue of clinical investigation has only recently been followed and must be further explored.

In summary, I have tried to indicate three avenues for

clinical investigation of the functioning of auditory cortex which have shown some promise of extending our knowledge of these regions.

REFERENCES.

1. GOLDSTEIN, ROBERT; GOODMAN, ALLAN, and KING, ROBERT B.: Hearing and Speech in Infantile Hemiplegia Before and After Left Hemispherectomy. *Neurology*, Vol. 6, No. 12:869-875, Dec., 1956.

2. PENFIELD, WILDER, and JASPER, HERBERT: "Epilepsy and the Functional Anatomy of the Human Brain." Little, Brown & Co., Boston, Mass., 1954.

3. GOLDRING, SIDNEY; O'LEARY, JAMES L., and KING, ROBERT B.: Singly and Repetitively Evoked Potentials in Human Cerebral Cortex with DC Changes. *E.E.G. Clin. Neurophysiol.*, (In press).

PERCEPTIVE HEARING LOSS.*

HAROLD F. SCHUKNECHT,

Henry Ford Hospital,

Detroit, Mich.

In recent years much investigation has been directed toward the physical and bioelectric mechanisms by which the cochlea receives the sound stimulus and codes it into neural impulses. Our knowledge of the auditory physiology beyond the cochlea is less well known. Future research in this area will be concerned with the mechanisms by which neural patterns are decoded and integrated with established memory patterns and other information, to result in hearing as we experience it.

THE HUMAN AUDITORY CORTEX.

The human cerebral cortex has been mapped according to minor variations in the pattern of the grey matter.[1,2,3] The auditory cortex of man is located in the superior temporal gyrus along the sylvian fissure, which corresponds to the topographic areas 41, 42 and 22 of Brodmann (see Fig. 1).

*Perceptive hearing loss here refers to that type due to disorders of the auditory pathways and cortex which is in accordance with the recent terminology suggested by the Committee for Conservation of Hearing of the American Academy of Ophthalmology and Otolaryngology.

Campbell has further divided this area into an audito-sensory region (area 41, thought to be related mainly to auditory perception) and an audito-psychic region (areas 42 and 22, thought to be important for auditory memory). These concepts, however, do not justify thinking of these areas as discrete functional units.

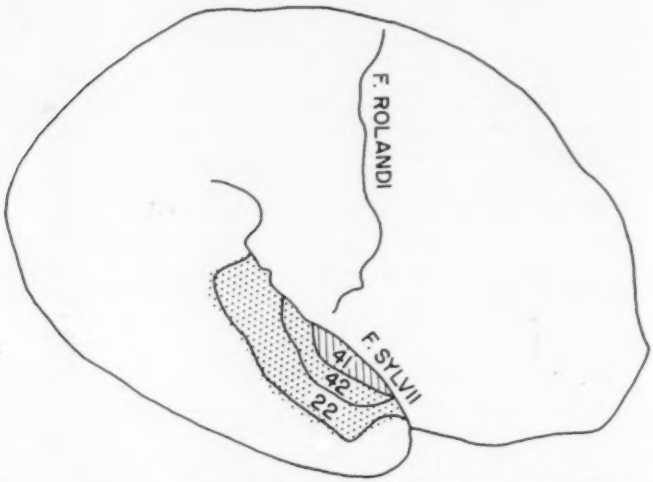


Fig. 1. Auditory cortex of man, as determined by cyto-architectonic studies. Shown are areas 41, 42 and 22 of Brodmann, and the audito-sensory (ruled) and audito-psychic (stippled) areas of Campbell.

Further evidence regarding the auditory cortex of man comes from the investigations of Penfield and Rasmussen,[4] who electrically stimulated the auditory cortex of humans during the course of intracranial operations. Local anesthesia was used, and the patients were asked to relate their auditory experiences.

Some of the sensations reported by the patients are listed below:

Ringing sound like doorbell.
Humming sound.
Low-pitched sound like a motor.

Continuous "clicking," like crickets at night.
Change in the sound of your voice.
Rushing sound, like a bird flying.
Contralateral ear felt as though she could not hear out of it.
Noise like birds chirping.
I hear funny things.
I hear "boom boom" on something.
Buzzing in contralateral ear.
Key of your voice changed.
I am a little deaf.

The records from these patients did not lend themselves to statistical study or detailed analysis; however, the investigators made the following observations:

1. Auditory responses were more numerous in the audito-psychic area of Campbell (areas 42 and 22 of Brodmann), and less frequent in the audito-sensory area (area 41 of Brodmann).

2. Electrical stimulation of the temporal lobe resulted in the sensation of sound in some patients and the sensation of being deaf in others; therefore, stimulation seemed to paralyze as well as activate.

3. The auditory sensations were most often referred to the contralateral ear.

4. Electrical stimulation close to the margins of the sylvian fissure were more apt to produce simple tones, ringing, etc., while stimulation away from the fissure was more apt to introduce an element of interpretation of the sound.

They also made a very interesting observation in patients having psychical seizures, the latter being described as a purely psychical state characterized by sudden change in perception of environment; a sort of dream state. Several patients having visual or auditory hallucinations in their psychic state experienced these phenomena when the temporal cortex was electrically stimulated. Apparently in these patients the cortex previously had been activated or conditioned by recurrent epileptic discharges, so that electrical stimula-

tion resulted in activation of acquired patterns of neural flow; in other words, the memory pathways.

LESIONS OF THE TEMPORAL LOBE.

The possible effect of unilateral temporal lobe tumors on hearing has interested clinicians for some time, but because hearing tests were lacking in quantitative values no consistent pattern of auditory loss was established for temporal lobe lesions.[5,5,7,8] Although bilateral temporal lobe lesions are less common, they can exist in Pick's disease and in cerebrovascular disorders, and in such cases the observers have consistently reported a total bilateral hearing loss.[5,9,10,11] With the development of electronic equipment, which permitted standardized audiometric evaluations, many new psychoacoustic phenomena have become manifest. One of the phenomena pertinent to this discussion only recently established is the loss of auditory intelligibility which accompanies temporal lobe tumor. This observation was slow in developing because the discrimination defect did not register in commonly used discrimination tests but became manifest only when a more difficult test was imposed upon the auditory system.

Apparently the amount of auditory information which is provided with each phoneme in normal speech is in excess to that which is necessary for intelligibility and, therefore, mild degrees of receptive aphasia (loss of auditory intelligibility) can exist without being detected by standard discrimination tests. Bocca, et al.,[12] and Pietrantoni, et al.,[13] have shown, however, that if a patient with a temporal lobe tumor is given a more difficult discrimination test, consisting of distorted speech (accelerating, interrupting, limiting frequency range, delivering in noise), the intelligibility is decreased in the ear contralateral to the temporal lobe lesion. Goldstein, et al.,[14] also have observed this phenomenon in patients with infantile hemiplegia.

Patients so afflicted are unable to understand the spoken language in spite of normal auditory acuity. Sounds reaching the ears of these patients are transmitted along the auditory pathways, but have no meaning. Perceptive aphasia

appears to be a true entity and is thought to result from aplasia, mal-development or trauma to the auditory cortex. Further proof is needed, however, to establish firmly the concept that auditory aphasia is the manifestation of a temporal lobe cortical deficit. A similar disorder, termed auditory imperception, is characterized by an ability to understand speech and develop language with auditory training, but difficulty in interpreting auditory tests involving discriminatory responses.[15] Derbyshire, et al.,[16] have shown that under favorable conditions auditory responses can be recorded in electroencephalograms of humans, and that the method can be used as an objective test of hearing. According to Rosenblith[17] it may be possible to develop that method into a practical clinical tool through the use of an electronic computer, which will sift the auditory response out of random background cortical activity. A serious limitation of objective auditory tests, of course, is that they do not tell us whether the responses reflect useful organized auditory information.

DECREASE IN NEURONE POPULATION.

Neff and associates[18] have shown that after bilateral ablation of the auditory cortex in cats, the pure-tone threshold is preserved; but more complicated discriminations, such as localization of sound and pitch discrimination, appear to be altered. The combination of circumstances in which there is a discrimination loss out of proportion to the change in pure-tone thresholds is not unique for cortical lesions, for it is also present when the first order neurone is partially destroyed by acoustic neurinoma, as was first pointed out by Woellner and myself in 1953,[19] and subsequently by Liden[20] and Walsh and Goodman[21] and Goodman.[22] (see Fig. 2).

The deafness of aging (prebycusis) also is often characterized by discrimination loss out of proportion to the pure-tone threshold loss (see Fig. 3). It has already been shown that the presbycusic ear often suffers greater degeneration of the cochlear nerve than of the corresponding organ of Corti[23,24,25] (see Fig. 4). In the aged person the neurone population at higher levels in the auditory system may also be decreased,

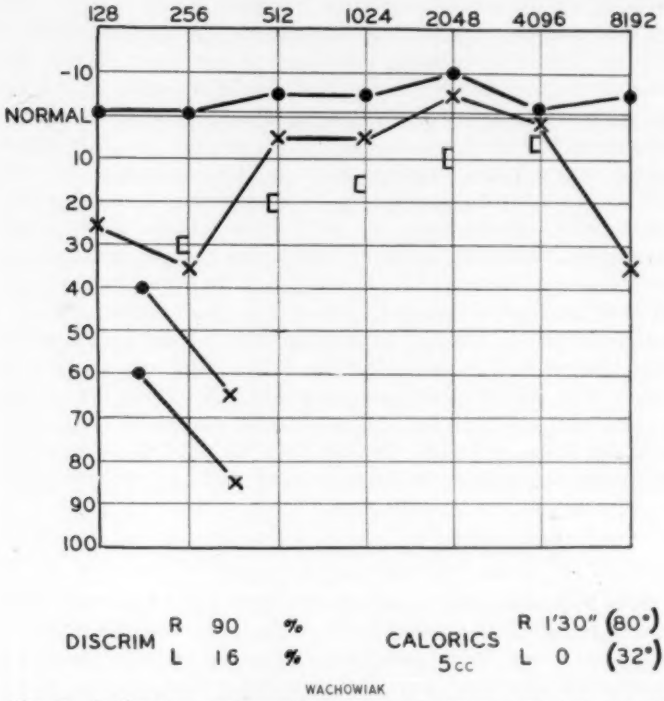


Fig. 2. Audiometric and vestibular tests of patient with left acoustic neurinoma proven subsequently at operation. In the left ear the pure-tone thresholds for the three speech frequencies (500, 1000 and 2000) were near normal; however, the speech discrimination score was only 16 per cent on repeated tests (PB-Max. Rush Hughes recorded word list).

for it is well established that the aging brain suffers a diffuse neuronal degeneration.

The auditory manifestations in patients with temporal lobe lesions, acoustic neurinoma and presbycusis, suggest that lesions involving the auditory pathways result in a system that transmits simple signals of threshold magnitude (such as pure tones) but cannot handle the complex signals of speech.

Woellner and I designed an experiment in which we further tested the concept that partial loss of fibers at the level of

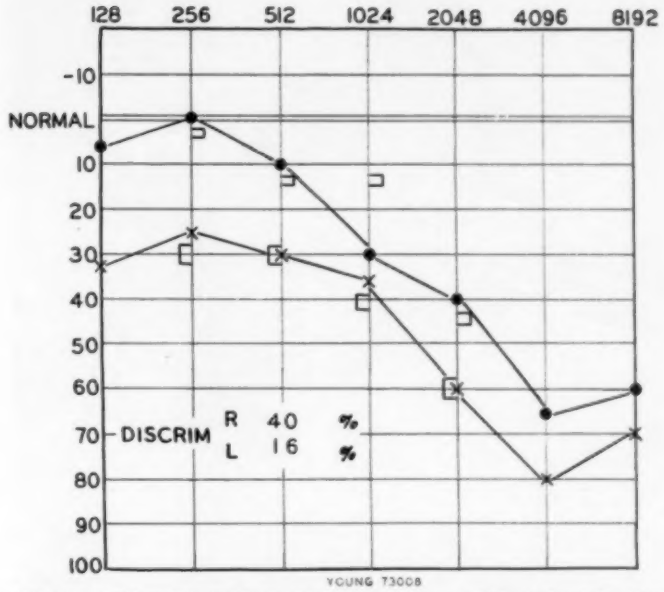


Fig. 3. Audiometric findings of 70-year-old man with presbycusis, showing discrimination loss out of proportion to loss for pure tones (PB-Max. live monitored voice).

the first order neurone is consistent with normal pure-tone thresholds. Working with conditioned cats we found that 75 per cent of the acoustic nerve fibers could be lost by actual count without creating a hearing loss for pure tones[26] (see Fig. 5).

COMMUNICATION THEORY.

In recent years concepts of information-transmission have been expressed in precise mathematical terms. Although few of us are capable of understanding the mathematics involved, we readily appreciate its importance for auditory intelligibility; the ear analyses speech by recoding the sound signals into impulses which travel up the auditory nerve. We know that the fundamental coding mechanism is very simple, and that a nerve impulse is either present or absent.

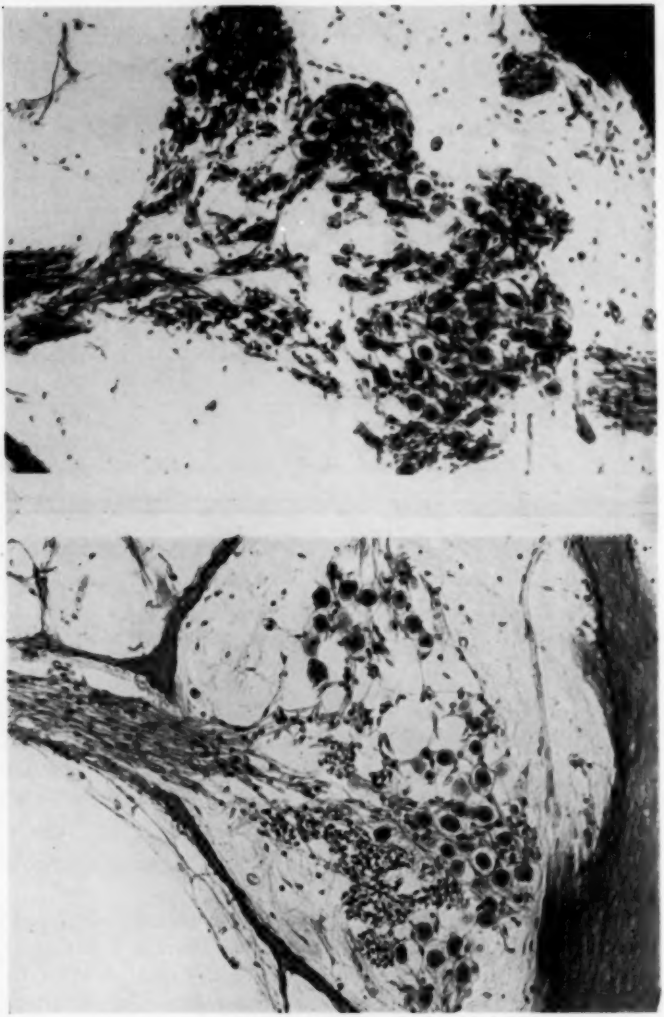


Fig. 4. Photomicrographs of spiral ganglion of normal young person (above), and 70-year-old man (below); both views from the upper basal turn. The aged man had severe degeneration of the organ of Corti and spiral ganglion in the lower basal turn, accounting for a high tone deafness. In the upper basal turn the organ of Corti was normal, but there was a loss of 30 per cent to 50 per cent of the spiral ganglia. It is this loss of neurone population which probably accounts for the deteriorating auditory discrimination (phonemic regression) in the aged.

This principle of "all or none" is basic to nerve physiology. Other limitations are that a single nerve fiber will carry a maximum of only 800 to 1000 impulses per second. We may enumerate the variables in a neurone pattern as follows: *1.* Frequency of impulses in a single fiber (below 1000 c.p.s.); *2.* time relation between impulses in the various fibers; *3.* selectivity as to which fibers are being activated; *4.* the num-

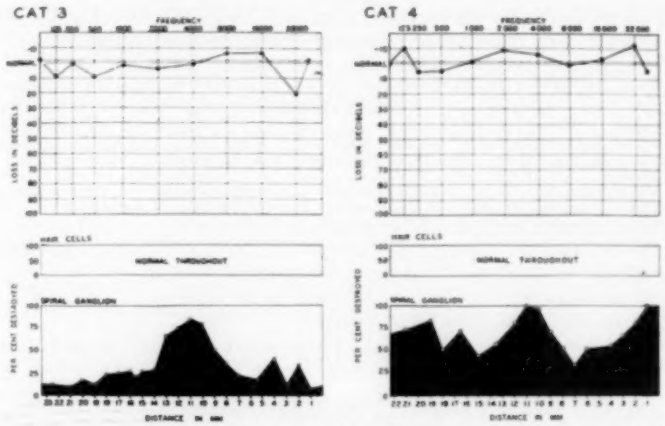


Fig. 5. Behavioral audiograms and cochlear charts for two animals with partial section of the cochlear nerve. The frequencies are placed along the abscissa of the audiogram in accordance with their anatomical distribution along the cochlear duct. The abscissa of the cochlear chart indicates millimeters along the cochlear duct as measured from the basal end, and the black filling shows per cent of ganglion cells lost as determined by actual count. Note that for Cat 2 there is an average loss of about 75 per cent of ganglion cells, but pure-tone thresholds remained normal.

ber of fibers being activated. In spite of the "all or none" law, the cochlear nerve can carry all of the information required for intelligibility of the spoken voice. This is possible because of the tremendous number of channels in this communication system, for we know there are about 35,000 channels, or fibers, in the human cochlear nerve. In a pathological auditory pathway with a loss of nerve fibers, a limitation is imposed upon the coding mechanism, and the amount of information which can be transmitted from the ear to the brain is less than normal.

CONCLUSIONS.

No one has yet evolved a completely clear concept of the psycho-acoustic manifestations resulting from lesions of the auditory pathways and centers. It is already apparent, however, that such lesions result in losses of intelligibility, varying from the mildest, detected only by special discrimination tests, to severe auditory aphasia. I am certain, however, that further physiological experimentation and clinical study will provide a better basis for the regional and functional diagnosis of disorders involving the auditory pathways.

REFERENCES.

1. CAMPBELL, A. W.: "Histological Studies on the Localization of Cerebral Function." Cambridge University Press, Cambridge, 1905.

2. BRODMANN, K.: "Vergleichende Lokalisationslehre der Grosshirnrinde in ihren Prinzipien Dargestellt auf Grand des Zellenbaues." J. A. Barth, Leipzig, 1909; reprinted 1925.

3. ECONOMO, C., and KOSKINAS, G. N.: "Die Cytoarchitektonik der Hirnrinde des erwachsenen Menschen." J. Springer, Vienna.

4. PENFIELD, W., and RASMUSSEN, T.: "The Cerebral Cortex of Man." The Macmillan Co., New York, 1955.

5. HENCHEN, I. E.: "Klinische und Anatomische Beitrage zur Pathologie des Gehirns." Stockholm, 1920.

6. SEIFERTH, L. B.: Verletzunger des Temporallappens und Otavusstörungen. *Arch. f. Ohren u.s.w. Heilkunde*, 155:357, 1949.

7. GRAHA: Ohr und Nervensystem. *Zbl. f. Neural. v. Psychol.*, 165:148, 1939.

8. MCNALLY, W.; ERICKSON, T. C.; SCOTT-MONCRIEFF, R., and REEVES, D. L.: Brain Tumors and Hearing. *Ann. Otol., Rhinol. and Laryngol.*, 45:797, 1936.

9. MOTT, F. W.: Bilateral Lesions of the Auditory Cortical Center; Complete Deafness and Aphasia. *Brit. Med. Jour.*, 310:1907 (Vol. 2).

10. BRAMWELL, E.: Case of Cortical Deafness. *Brain*, Oct., 1927.

11. PICK: Reference by Pietrantoni, et al.

12. BOCCA, E.; CALEARO, C.; CASSINARI, V., and MIGLIVACCA, F.: Testing Cortical Hearing in Temporal Lobe Tumors. *Acta Otol.*, 45:289, 1955.

13. PIETRANTONI, L.; BOCCA, E., and AGAZZI, C.: "Diagnose delle Sordita Retroccocleari Idos." Milano, 1956.

14. GOLDSTEIN, R.; GOODMAN, A., and KING, R.: Hearing and Speech in Infantile Hemiplegia Before and After Left Hemispherectomy. *Neurol.*, 6:869, 1956.

15. HARDY, W.: Personal communication.

16. DERBYSHIRE, A. G.; FRASER, A. A.; MCDERMOTT, M., and BRIDGE, A.: Audiometric Measurements by Electroencephalography. *E.E.G. and Clin. Neurophys.*, 8:467, 1956.

17. Rosenblith, W. A.: Relation Between Auditory Psycho-physics and Auditory Electro-physiology. *Trans. N. Y. Acad. Sci.*, 19:650, May, 1957.

18. Neff, W. D., and Yela, M.: Function of the Auditory Cortex: The Localization of Sound in Space. *Amer. Psychol.*, 3:243, 1948; Jerison, H., and Neff, W. D.: Effect of Cortical Ablation in the Monkey on Discrimination of Tonal Patterns. *Fed. Proc.*, 12:101, 1953.

19. Schuknecht, H. F., and Woellner, R. C.: Hearing Losses Following Partial Section of the Cochlear Nerve. The Laryngoscope, 63:441, 1953.

20. Liden, G.: Speech Audiometry. *Acta Otol.*, suppl., 114, Stockholm, 1954.

21. Walsh, T. E., and Goodman, A.: Speech Discrimination in Central Auditory Lesions. The Laryngoscope, 65:1, 1955.

22. Goodman, A. C.: Some Relations Between Auditory Function and Intracranial Lesions with Particular Reference to Lesions of the Cerebellopontine Angle. The Laryngoscope, 67:987, 1957.

23. Schuknecht, H. F.: Presbycusis. The Laryngoscope, 65:402, 1955.

24. Crowe, S. J.; Guild, S. R., and Polvogt, L. M.: Observations on the Pathology of High Tone Deafness. *Bull. Johns Hopkins Hosp.*, 54:315, 1934.

25. Saxen, A.: Pathologie und Klinik der Alterschwerhörigkeit. *Acta Otol.*, Suppl. 23, 1937; Inner Ear in Presbycusis. *Acta Otol.*, 41:213, 1952.

26. Schuknecht, H. F., and Woellner, R. C.: An Experimental and Clinical Study of Deafness from Lesions of the Cochlear Nerve. *Jour. Laryngol. and Otol.*, 69:75, 1955.

THURSDAY MORNING SESSION,

May 16, 1957.

HEARING LOSS AND NOISE EXPOSURE.

The International Conference on Audiology convened at 9:30 A.M., in the Auditorium of the Washington University School of Medicine, St. Louis, Missouri; Prof. Walter A. Rosenblith, Associate Professor of Communications Biophysics, Massachusetts Institute of Technology, Cambridge, Massachusetts, presiding.

DR. IRA J. HIRSH, (St. Louis, Missouri): Unfortunately, Dr. Kenneth Day could not be with us, and Prof. Walter A. Rosenblith has consented to act as chairman for this session.

NOISE IN INDUSTRY.

JEROME R. COX, JR.,

Central Institute for the Deaf,

St. Louis, Missouri.

This is a report on some noise measurements that were made in a number of factories in a certain section of the United States. These measurements were directed towards evaluating individual noise exposures. The goal of the survey was some statistical indication of the industrial noise exposures in a section of the country. Before presenting the procedure for the survey and the results, I will give a brief review of noise measurements in the United States.

In 1925, noise measurements were made in the City of New York, with the aid of the 3-A audiometer, an early Western Electric instrument. The masking produced by the noise was measured by the audiometer. The sound pressure levels were then calculated from the masking. This seems like an

ancient method now, but it was a popular technique then. About 1930 the first sound level meters were introduced. Because the users soon found that many sounds gave the same reading on the meters but did not sound the same, an effort was made to approximate the characteristics of the ear. This effort was unsuccessful, but the results of it remain with us as the A, B, and C scales of modern instruments.

It soon became clear that some sort of frequency analysis was necessary. Various narrow-band analyzers were developed, but their use was limited because of the tedious nature of the measurement. The more modern octave band analyzer strikes a happy medium between detailed frequency analysis and ease of measurement. It has been used extensively in the past decade, and most industrial noise data are today being reported in octave bands. Recently, fractional octave bands have appeared but do not, to date, enjoy widespread use because of the added complexity of reporting the data.

Until a few years ago sound pressure level and frequency were the primary parameters involved in noise measurement. All measurements were essentially cross-sections in time. Very little attention was paid to the fluctuations and variations of the noise as time passed. An excellent compilation of industrial noise measurements of this type has been prepared by Karplus and Bonvallet, *(American Industrial Hygiene Association Quarterly,* Vol. 14, page 235, 1953); however, measurements of this sort can only be related to impairment of hearing when the exposure is steady throughout the working day, and the noise has no unusual transient characteristics.

Since the relations of noise to hearing loss are of great importance in the field of industrial noise, more emphasis has been placed in recent years upon the design of instruments that give information about the transient character of the noises. The impact meter, developed by General Radio, and the exposure meter, developed by Crouch, are two examples of this trend toward instruments that are aimed at the measurement of the time-varying characteristics of noise. Research sponsored by the Armed Forces has also been di-

rected toward the measurement of noise exposure rather than the simple frequency distribution that was so popular during the past decade.

I think the concept of a noise exposure is one of the important developments in the field of industrial noise. It is usually assumed that time and intensity are equally important in determining the noise exposure. A quantitative way to state this assumption is to say that sound energy, the product of time and intensity, is proportional to the noise exposure, and, therefore, directly related to the hearing loss that may result from the exposure. Students of physics will recall that the product of time and intensity is energy—in this case sound energy.

In computing sound energy, intensity is expressed in watts per unit area rather than in decibels, a logarithmic unit. Translated into decibels, the energy rule means that a noise present throughout an eight-hour working day would be equivalent to a similar noise that is present during one-half the working day, but three decibels more intense. Similarly, a noise present only one-tenth of the working day is equivalent to a noise ten decibels less intense, but present throughout the working day. The assumption that noise exposure is proportional to the sound energy received by the ear has not been adequately validated. It is a frequent assumption, and I will make no attempt to justify it.

In order to evaluate noise exposure a tremendous amount of data is necessary. It is almost unbelievable to people who have not encountered the problem. Data must be taken on the intensity, frequency distribution, transient characteristics, and the daily time pattern of the noise. In addition, the workers' movements in the noisy area, and the characteristics of the sound field, must be recorded. With these data, it is possible to estimate the noise exposure according to the assumption that exposure is proportional to sound energy. In the measurements to be reported, we attempted to gather all of the information necessary for the evaluation of every man's noise exposure in each of the plants surveyed.

A general data sheet gave information about the kind of

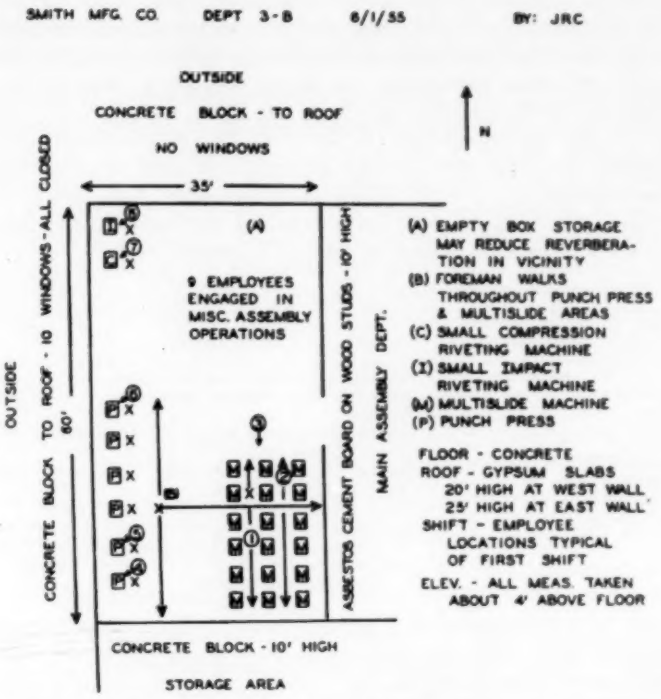

SMITH MFG. CO. DEPT 3-B 8/1/55 BY: JRC
OUTSIDE
CONCRETE BLOCK - TO ROOF
NO WINDOWS
35'
N
(A)
9 EMPLOYEES ENGAGED IN MISC. ASSEMBLY OPERATIONS
OUTSIDE
CONCRETE BLOCK TO ROOF - 10 WINDOWS - ALL CLOSED
60'
ASBESTOS CEMENT BOARD ON WOOD STUDS - 10' HIGH
MAIN ASSEMBLY DEPT.
(A) EMPTY BOX STORAGE MAY REDUCE REVERBERATION IN VICINITY
(B) FOREMAN WALKS THROUGHOUT PUNCH PRESS & MULTISLIDE AREAS
(C) SMALL COMPRESSION RIVETING MACHINE
(I) SMALL IMPACT RIVETING MACHINE
(M) MULTISLIDE MACHINE
(P) PUNCH PRESS
FLOOR - CONCRETE
ROOF - GYPSUM SLABS 20' HIGH AT WEST WALL 25' HIGH AT EAST WALL
SHIFT - EMPLOYEE LOCATIONS TYPICAL OF FIRST SHIFT
ELEV. - ALL MEAS. TAKEN ABOUT 4' ABOVE FLOOR
CONCRETE BLOCK - 10' HIGH
STORAGE AREA

Fig. 1. Typical floor plan of department surveyed. All significant noise sources in this small shop are identified, working areas of employees are indicated by crosses, and each measuring station is marked by a circled number.

industry and the number of employees, shifts, breaks in the daily exposure, and other departmental information. The fundamental framework for reporting our data is shown in Fig. 1.

This is a drawing typical of the departments surveyed. This is the floor plan of a small shop, showing all the significant noise sources, the location of all the workers, the location of each of the measuring stations, and, in addition, information about the acoustical environment and movements of employees who do not remain in a fixed position.

Noise measurements were sometimes made with octave bands, but often a simple sound survey meter sufficed because it is faster, less conspicuous, and satisfactory for rough evaluations of noise exposures. A rough frequency analysis can be made from readings taken on the three scales, A, B, and C, of the survey meter (J. R. Cox, Jr., Approximate frequency analysis from weighting-network data in acoustic measurements. *General Radio Experimenter*, Vol. 28, p. 1, 1953). This scheme worked because of the statistical nature of the survey, but cannot be generally recommended.

Location No.	Meter Readings(db.) A B C	REMARKS
8	98 98 98	1. A small riveting machine. Connector is held on anvil and hammer sets rivet. Repeated impact. 2. About 300 blows/min. Operator makes 1-2 parts/min. 3. Sound of hammer striking small part. No other source of noise. 4. Each rivet takes 5-10 sec. 4 bursts/part. Machine in use when compression riveting is behind schedule. Foreman estimates that this occurs about 25% of the time. Operator is otherwise unexposed.

Fig. 2. A small section of the form used to report data from each of the measurements stations. The four items at the right describe the noisy machine, its operating conditions, the nature of the noise and the noise exposure pattern. The number at the left refers to the location of the measurement shown on Fig. 1.

Figure 2 shows a small section of the form used to report data from each measurement station. At the right we see four items: the nature of the noisy machine, the operating conditions of the machine, the nature of the noise from the machine in terms of some more familiar noise, and the noise exposure pattern. In this example the machine is on only about twenty-five per cent of the time.

The survey covered a total of seventy-five plants in twelve different classifications used by the Census Bureau. Of the remaining eight, several were not noisy industries, some employ very few people, and one was not found in the area in which this survey was conducted; therefore, it is believed that a sufficiently large variety of noisy industries

was studied. The total number of workers whose exposures were evaluated is just slightly under thirty thousand. As you can well imagine, considerable data was accumulated.

Because of the scope of the survey, it was necessary that a simple scheme be developed for analysis of the data. The following procedure was followed: *1.* A rough frequency analysis was carried out to estimate the sound pressure level in the 300-600 c.p.s. band. This octave was chosen because it is the important sorting octave, according to the data presented in the report of Committee Z.24-X-2 of the American Standards Association (American Standards Association, *The Relations of Hearing Loss to Noise Exposure,* Z.24-X-2 Report. New York, 1954). *2.* The equivalent exposure level was found by using the assumption that energy is proportional to exposure discussed a few moments ago. *3.* Appropriate adjustments based on experience with similar machines in other plants must be employed, because quite often data are not as complete as they should be. *4.* All of the noise exposures are categorized into the four classifications shown in Fig. 3.

Classification of Noise Exposure	A	B	C	D
Equivalent Sound Pressure Level in the 300-600 c.p.s Octave Band.	Greater Than 105 db.	105 db. to 95 db.	95 db. to 85 db.	Less Than 85 db.

Fig. 3. Classification of noise exposures according to the steady sound pressure level having a total energy each day equal to that in the 300-600 c.p.s. octave of the actual noise.

The equivalent sound pressure level shown is in the 300-600 c.p.s. octave band. All of the noise exposures are sorted into these four classifications. After obtaining the totals in each classification we were surprised to find fewer A exposures than anticipated. Upon re-examining the distribution of industries in the area studied we find a heavy representation of industries having only a few A exposures; therefore, an adjustment of each of the major classifications of industry was made on the basis of the census of manufacturers.

The total number of workers in noisy industries is shown in Fig. 4. The population of the nation at the time of the survey was a hundred and sixty million. The total number of people not in schools or hospitals is one hundred and ten million. The labor force is sixty-three million, but the labor force in the manufacturing industries is only seventeen million. Excluding administrative workers the remainder is fourteen million, of whom only ten million are in industries

	Millions
U. S. Population (1953)	160
Non-Institutional Population	110
Labor Force	63
Labor Force in Manufacturing	17
Production Workers in Manufacturing	14
Production Workers in Noisy Industries	10

Fig. 4. Distribution of the U. S. population at the time of the survey. Noisy industries exclude those that generally have no significant noise exposures anywhere in the industry, i.e. the apparel industry.

PERCENTAGE OF PRODUCTION WORKERS IN NOISY INDUSTRY IN VARIOUS NOISE EXPOSURE CLASSIFICATIONS.

	A	B	C	D
Percentage	0.5%	4.5%	17%	78%

Fig. 5. Estimate of the percentage of workers in noisy industries who are exposed to various noise exposure classifications as defined in Fig. 3. Percentages are adjusted from regional survey data to reflect national distribution of noise exposures. The Armed Services, transportation and construction workers and farmers have been excluded from the survey.

that have significant noise exposures. It is estimated that those ten million workers are distributed among the noise exposures A, B, C, and D, according to the percentages shown in Fig. 5.

One is surprised that the percentage in the A classification is so small. Remember, however, that the Armed Services, transportation and construction workers, and farmers have been excluded from the study. Only workers in manufacturing industries that have significant noise exposures have been included.

In conclusion, there are a few severe noise exposures, but many less severe ones; in fact, if all of the workers in the

nation subjected to A exposures were suddenly to incur a 50 db. hearing loss, the mean threshold hearing level of the nation would shift by only 0.2 db. This verifies the fact that there must be many other causes of hearing loss. There are others not considered in this study who have serious noise exposures. Many people use firearms extensively in and out of the Armed Services. In addition, there are other kinds of hearing losses that are difficult to differentiate from hearing loss caused by noise.

One final statement that deserves emphasis is: just because a man works in industry is not a sufficient reason to assume that his hearing loss is caused by noise.

HEARING LOSS IN INDUSTRY.

ARAM GLORIG,
WILLIAM GRINGS,
ANNE SUMMERFIELD,
Subcommittee on Noise in Industry,
Los Angeles, Calif.

INTRODUCTION.

Valid information about the relations of hearing loss to noise-exposure is difficult to obtain. Undoubtedly, this difficulty is due in part to the complexity of the relations, but there are also many obstacles that must be overcome before we can obtain suitable data from which to draw valid conclusions about these complex relations. It is the purpose of this paper to discuss a recent study of industrial hearing loss as a means of showing some of the difficulties encountered in making inferences from existing audiometric records, to discuss the difficulties generally encountered in field research in hearing, and to describe our attempts to lessen these difficulties.

Two important questions to be answered are: *1.* How much hearing loss results from noise-exposure; *2.* what is the rela-

tive importance of different conditions that contribute to the production of hearing loss? To answer these questions we must have measurements of many noise-exposures and serial audiograms of persons exposed to each noise; serial audiograms consist of an original and several well spaced repeat audiograms. The audiometric measurements must be accurate, and the information contained in the description of the noise-exposure must be complete and detailed.

The ideal approach to the problem of hearing loss in industry would be to carry out controlled experiments in industrial plants. For these experiments we would require ideal testing environments and complete freedom for scheduling tests. We would test many persons at repeated intervals of various lengths. Different groups of these persons would be exposed to noise of different specific characteristics, and there would be no overlapping of the specific exposures. Obviously, practical considerations limit the achievement of such ideal experimental conditions in the field. It has been possible, through the generous cooperation of certain companies, to do controlled experiments in the field with small numbers of persons, but for some of the statistical analyses we wish to perform we need many thousands of audiograms. It is not possible to collect this special data from large groups of persons in the field. We are restricted to the information we can obtain from the data records that are made available to us from hearing conservation programs already organized and carried out in industry. As a specific illustration of this type of data analysis, let me describe a recent study made at the Research Center of the Subcommittee on Noise in Industry.

RECENT STUDY OF INDUSTRIAL HEARING LOSS.

The raw data for this study were obtained from records which had been made available by a large aircraft manufacturer. These data were entered on punched cards and were then analyzed with respect to three topics: *1.* Evidence for hearing loss caused by noise-exposure; *2.* differences in hearing loss occurring in different plant occupations within the company; and *3.* possible sources of error which might invalidate the results of the study.

The data sampled contains 9,145 audiograms of industrial and flight line personnel. The audiograms represented 5,816 different individuals—3,212 factory workers and 2,604 flight line personnel. There were 3,229 repeat audiograms. The repeat audiograms included both periodic recheck and special medical referrals, and it was not possible from the record to differentiate between these two.

Supplementary information provided with the audiograms included age, history of exposure to noise, job category and medical history. Age was indicated by groups of years only; 18-25 years, 26-35 years, 36-45 years and 46 years or over. Exposure to factory noise was recorded in three classes: less than one year, 1-5 years and 5 years and over. Flight line (jet) exposure time was specified in hours: 0-100, 100-500, and 500 hours or over. Most persons had work experience in more than one job category. The largest "pure" work group, 1,196, was made up of riveters. Accurate serial records of noise-exposure factors and of information about changes in medical or work history were not always available.

The data contained evidence of the production of hearing loss by noise-exposure. Fig. 1 shows curves of hearing loss vs. years of noise-exposure for groups of male factory workers. Separate curves are presented for each of three age groups: 18-25, 26-35 and 36-45 years. Note that the curves show increasing high frequency hearing loss with increasing exposure time. At frequencies below 2000 c.p.s. there is less evidence of systematic change. Hearing loss as a function of years of exposure for 1,190 riveters is plotted in Fig. 2. According to the information available in the records, these men had been exposed to riveting noise only. Note that the hearing losses shown by these men are very similar to the losses displayed in Fig. 1, which represents the entire group of factory workers. Tests of statistical significance were applied to these data, and the results indicate that hearing losses at frequencies above 3000 c.p.s. are statistically significant. At 3000 c.p.s., losses in the older age group only are statistically significant, and below 3000 c.p.s. the indicated losses are statistically not significant. From these results we would infer that on the average, as the years in noise increase so

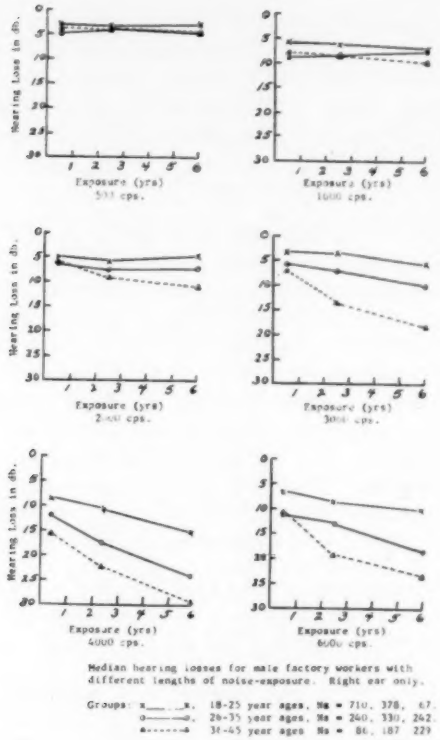
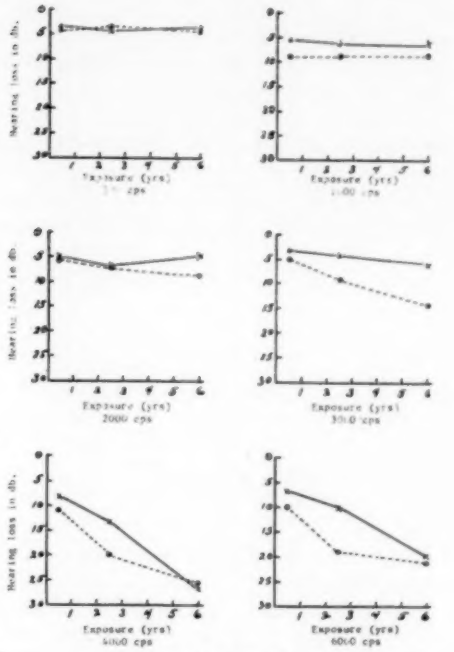

Hearing Loss in db.
Exposure (yrs)
4000 cps.
6000 cps.
Median hearing losses for male factory workers with different lengths of noise-exposure. Right ear only.
Groups: x——x, 18-25 year ages, Ns = 710, 378, 67.
o——o, 26-35 year ages, Ns = 240, 330, 242.
Δ------Δ 36-45 year ages Ns = 86, 187 229

Fig. 1.

does hearing loss increase at tested frequencies above 3000 c.p.s.

The data were also analyzed with respect to differences in hearing loss that might occur in different occupations in the aircraft company. A comparison of the hearing losses of riveters and of jet flight line mechanics may be made from the curves presented in Fig. 3. Note that there is a consistent difference between the median losses of the two groups, but that the flight line mechanics have the better hearing in all age groups. The differences are probably statistically significant, but tests for statistical significance were not made because of



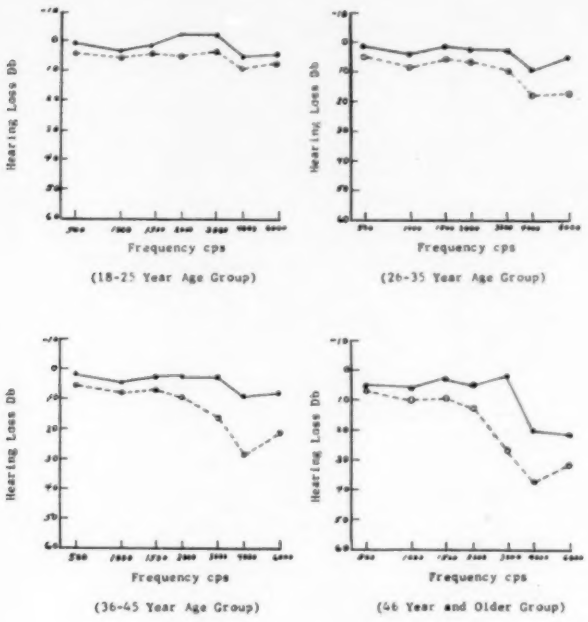
Median hearing loss for 1190 workers with riveting experience only, with different lengths of exposure. Right ear only.

Groups: X——X, 18-25 year ages, Ns = 604, 161, 20.
O-----O, 26-35 year ages, Ns = 169, 143, 93.

Fig. 2.

some disconcerting and unexplained discrepancies which appeared when the curves were compared with hearing loss curves from other studies.

Specifically, the average hearing losses at the higher frequencies for both groups from the aircraft plant, (riveters and flight line mechanics) are less than hearing losses for *any* occupational group, even for office workers, measured at a State Fair Hearing Survey conducted at Milwaukee, Wisc., in 1954. This result suggests that a search should be made for possible sources of bias in both sets of data. Some unspecified selective factors may have operated in the assign-



Comparison of median hearing losses for men with riveting experience and with jet engine flight line exposure. Right ear only.

Groups: ●——● Jet engine flight line exposure
○- - -○ Riveting

Fig. 3.

ment of men to a particular work category in the aircraft plant; then too, the audiograms in this plant may have been influenced by certain medical referral policies.

The records with the serial audiograms were checked carefully for obvious errors such as decreasing age, impossible changes in years of noise-exposure such as a history of five years in noise on one record and six months later a history of seven years in noise, etc. These data showed errors of this type in less than 1 per cent of the records.

What then have we learned from this analysis? The data

indicate that in general, high frequency hearing loss increases with increase in noise-exposure time up to six years, the time period for which data were available; that within the company jet flight line mechanics show less loss than riveters, and that these riveters and mechanics show less hearing loss than do comparable age groups in another study of men in what are considered to be *non-noisy* occupations. Findings such as the latter are unexplained at present, and we are understandably reluctant to accept at face value, or to publish without explanation, the results of this and other similar studies.

Our experience, as exemplified in the study above, has indicated that many of the differences between groups of data are not strictly related to differences in noise-exposures, or even to such relevant factors as age. Before we can draw valid conclusions about the relation of hearing loss to noise-exposure, we must find ways to minimize the importance of, or to control statistically the factors that are not related to the noise-exposure. The remainder of this paper will be devoted to a discussion of some of the difficulties encountered in gathering and analyzing data from field studies of noise-induced hearing loss and to a brief description of attempts that are being made to effect the necessary controls.

EXPERIMENTAL DIFFICULTIES.

In the main, the difficulties encountered in our field research in industry are of two kinds: those peculiar to studies of noise-induced hearing loss and those common to all experiments. Three of the problems peculiar to studies of noise-induced hearing loss are:

1. Description of Noise-exposure. One of the most difficult problems associated with a study of noise-exposure is that of devising a method of describing and categorizing in a meaningful way the multitudinous types of noise-exposure encountered. In order to describe noise-exposure adequately for correlation purposes, some numerical method of classification must be evolved. This method of classification must provide a way of combining the measurement of quantity of sound

energy with measurements of its time distribution. The result might be called a "noise-exposure-index." One approach to the total problem would be to index noise-exposure first with respect to its *physical dimensions,* and then as a further step, to index the effects of the noise-exposure on man. The problem of assessing noise-exposure is very complex, and large amounts of data must be gathered and analyzed before we can say what a practical and useful assessment of noise-exposure is.

Recent measurements indicate that sufficiently detailed information about the energy content of noise-exposure is not provided by RMS measurements. A report from Harold Crouch, of Eastman Kodak Co., shows that although the RMS values of overall levels of certain so-called "steady state" noises were in the region of 100 db., the instantaneous levels were as high as 120 db. for 10 per cent or 15 per cent of the time. In my opinion, evidence such as this must not be overlooked. We may find that peak factors in noise-exposure correlate better with resulting hearing loss than do some of the factors we have already considered, such as the frequency distribution of energy in the noise-exposure. In spite of the variability of the industrial processes and of the accompanying noise-exposures, large numbers of industrial audiograms show the same type of hearing loss: the loss begins in the region of 4000 c.p.s. and spreads in both directions as exposure time increases. It appears then, that energy content of noise, its peaks, its time distribution and its total duration, regardless of spectral distribution, are factors which bear close scrutiny.

Although we are cognizant of the fact that time plays a part in the production of hearing loss, we have not yet learned what time interval is most suitable for correlation purposes; should it be one work day, one work year, or some point between?

The Research Center of the Subcommittee on Noise in Industry has recently undertaken a program of gathering and analyzing detailed information about noise-exposures. The first goal of this program is to devise a method of indexing

noise-exposures from measurements of their physical parameters; the second goal, which I fear will not be realized for some years, will be to index the auditory effects of various noise-exposures on man.

2. Temporary Threshold Shift. A second difficulty peculiar to studies of hearing loss is caused by the temporary hearing loss that results from several hours exposure to noise. Recovery from this loss is usually complete a few hours after cessation of exposure, with the major part of the recovery normally occurring in the first 30 to 60 minutes. The existence of this temporary loss, or *temporary threshold shift*, has an important influence on the validity of field data. Recent measurements made in our laboratory indicate that at 4000 c.p.s. the temporary threshold shift resulting from two hours' exposure to white noise may be less than 5 db. in one person and more than 35 to 40 db. in another. Fig. 4 shows the mean threshold shift of a group of ten subjects whose hearing was measured at half-hour intervals, during and after a two-hour exposure to white noise of 100 db. overall sound pressure level. There appears to be no systematic variation in the size of the shift experienced by different individuals.

We have also made field measurements of the temporary threshold shift caused by exposure to typical industrial noises. These measurements were made throughout the workday on several hundred employees of an aircraft company. The field findings confirmed the laboratory findings.

If industrial hearing tests are made late in the workday after employees have experienced several hours of exposure to noise, the resulting audiograms will be contaminated by temporary threshold shifts. Temporary threshold shift, as a function of the time of day the audiogram is made, can cause a considerable variation in an individual's audiograms; therefore, if some correction for temporary threshold shift is not applied to industrial audiograms the true threshold of hearing will be disguised by temporary threshold shifts, and much of the available field data will not be suitable for studies of permanent hearing loss.

In what ways can we control the effect of temporary threshold shift? Ideally, we might use only those audiograms that were made at least 16 hours after cessation of exposure to noise. This obviously limits us to too few audiograms for good statistical analyses. On the other hand, it is impossible for industry to make all audiograms at the beginning of the

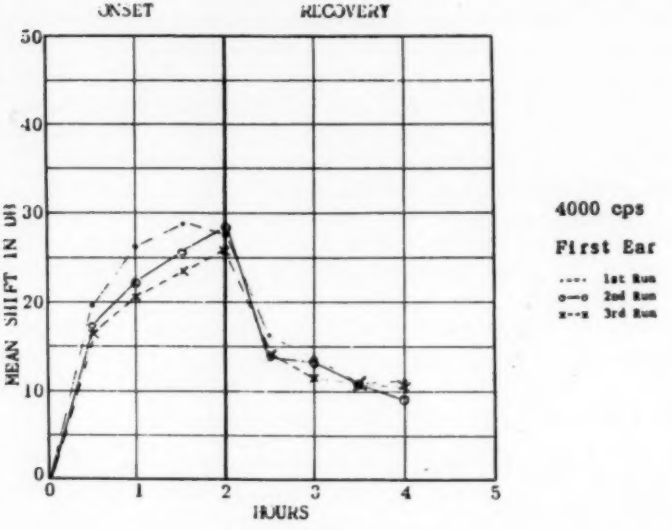


Fig. 4.

workday before employees have been exposed to noise. Our most fruitful approach seems to be to devise some valid statistical method for controlling the effect of temporary threshold shift. We hope to gain enough information from our laboratory studies and from analyses of large numbers of industrial records which include the time of day the audiogram was made to be able to devise such a method. The laboratory measurements will tell us, for continuous exposure to noises which are similar to white noise, how much temporary threshold shift is to be expected on the average at different hours during the work day. Incidentally, it should

be made clear that recording the time of day the audiogram was made is not sufficient; a statement which describes the amount of noise-exposure prior to the time of giving the hearing test should also be included. Further, there are promising indications from our studies of industrial records that if we have a measure of the noise-exposure we may be able to use late-in-the-day audiograms to predict the average "pre-exposure" threshold for a group of employees, or as I prefer to call it, the average *resting* threshold for the group.

3. Hearing loss that accompanies age. A third difficulty which besets the study of noise-induced hearing loss is the compounding of inner-ear hearing losses due to several causes. Age, otological disease and noise-exposure may all produce inner-ear hearing loss. There is no way of telling from the individual audiogram which of these causes produced a given loss. Assuming that we can identify all cases of otological disease from medical histories and so can eliminate them from samples to be studied, we are still faced with the problem of separating noise-induced hearing loss from "age-induced" hearing loss.

There is a semantic difficulty here as well as a procedural problem. Let me explain. The word most commonly used to denote loss of hearing with age in "presbycusis." Strictly speaking, "presbycusis" denotes only that there is a relation between hearing loss and age; it does not specify what the relation is. The meaning of the word has become somewhat restricted, however, by usage and by analogy with the restricted meaning of other terms containing the prefix "presby-." In medical literature, the word "presbycusis" has been used commonly to denote hearing loss that accompanies age and that results only from physiological changes that occur with the passage of time. Schuknecht* describes the cause of presbycusis as "degenerative changes beginning at the basal end of the cochlea and extending toward the apex, affecting almost equally and simultaneously the various structures within the cochlear duct, including the afferent and efferent

*Schuknecht, H. F.: Prebycusis. The Laryngoscope, 45:402-419, June, 1955.

nerve fibers. The formation of this atrophic lesion resembles the age process known to affect other tissues as age increases."

Although the term "presbycusis" is not now so restricted, we feel that it should be reserved to denote specifically the hearing loss that is produced as a result of the aging process, and that is not directly related to environmental factors. In addition to the term "prebycusis," then, we need words to describe inner-ear hearing losses caused by noise-exposures. The large hearing losses of noise workers certainly cannot be considered as a true reflection of changes produced by age alone; in part, these losses are caused directly by the environment. There is also some indication that a large proportion of the population, including persons not employed in noisy occupations, may sustain an average noise-induced hearing loss that is greater than presbycusis but is considerably less than the average noise-induced occupational loss. This smaller noise-induced loss results presumably from exposure to social and domestic noises that are common in our society. We are now making measurements to determine the magnitude of this loss.

At this time we consider inner-ear hearing losses not caused by otological disease to be made up of one or more of these three losses:

1. The loss produced by physiological aging processes, or "prebycusis." Presbycusis has no direct causal relation to noise-exposure.

2. The loss produced by noise-exposures not greater than those found in ordinary environmental social situations. For want of a better term this hearing loss might be called "sociocusis." Sociocusis is causally related to the noise-exposure encountered in non-occupational pursuits and/or in occupations where the noise levels do not ordinarily exceed those found in our social environments. The noise-exposures in most offices, for example, are less than the exposures one encounters in many social and domestic activities such as riding in an automobile with the windows open, operating washing machines, vacuum cleaners, electric mixers and other noise producing home appliances. These two losses, presby-

cusis and sociocusis, are not necessarily related to one's occupation. Disease, diet, stress and social environmental noise undoubtedly produce a gradual loss of hearing which increases with advancing age. This concept of "wear and tear" on the auditory system from non-occupational causes must be considered if we are to come to any sensible decision regarding noise-induced hearing loss.

3. The loss produced by industrial or military high level noise-exposures, or, if you will, "occupational hearing loss."

In order to draw conclusions regarding the auditory effects of specific noise-exposures we must provide a method for separating out the non-noise-exposure-related hearing loss. Our present concept is that occupational hearing loss and the hearing loss accompanying age are additive losses. The true nature of the interrelation of noise-exposure and other causes of hearing loss remains to be determined, but until it is, we shall continue to assume that inner-ear hearing losses, from whatever cause, are additive. Under this assumption we determine the average occupational hearing loss as follows: *1.* measure the total hearing loss, *2.* subtract from this measured hearing loss the amount of loss to be expected from non-occupational causes; the remainder is the occupational hearing loss. How large is the expected non-occupational loss? The answer may be found by measuring the hearing of many persons who have no otological disease and who have not been exposed to high noise levels for extended periods of time. The curves shown in Figs. 5 and 6 are representative of the "non-occupational hearing loss that accompanies age" for males and females respectively. These curves "summarize" the hearing measurements made on several hundred male office workers and female office workers, housewives, etc., with no history of disease or occupational noise-exposure.

FIELD EXPERIMENTS DIFFICULT TO CONTROL.

Let us now consider some of the difficulties associated with the actual gathering of data in the field.

First, from a practical standpoint the process of hearing measurement alone presents many almost insurmountable

difficulties. For research purposes we should have measurements of threshold responses to pure tone stimuli, but it is practically impossible to make such measurements for the employees in large industries. A trend toward screening audiometry is becoming evident in the larger plants. There is usually time for threshold tests during the pre-employment physical examinations, however, and we have consistently urged that threshold hearing tests be made a part of this

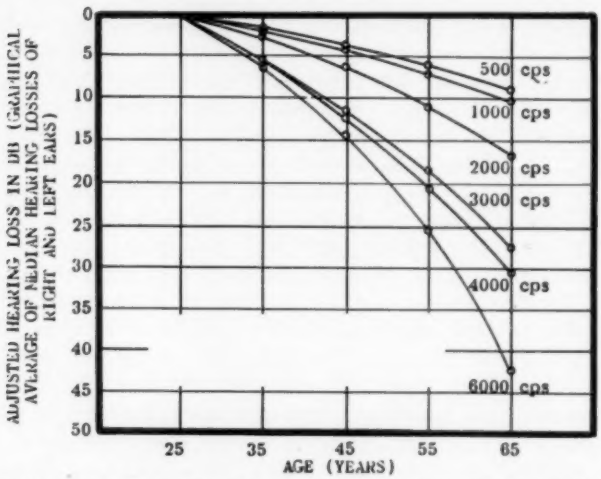


Fig. 5. Curves of presbycusis and sociocusis, (nonoccupational hearing loss that accompanies age) male office workers—1954 Wisconsin State Fair Hearing Survey (604 ears).

examination. We have also recommended that rapid follow-up tests of hearing or recheck tests be made for many employees with the single frequency screening method devised by Dr. Howard House and myself. The time saved by use of this screening technique should make it possible for most industries to give threshold tests to employees whose hearing has undergone a significant change. These recommendations and many others have been made available to industry in our revised Guide for Conservation of Hearing in Noise, published in 1957.

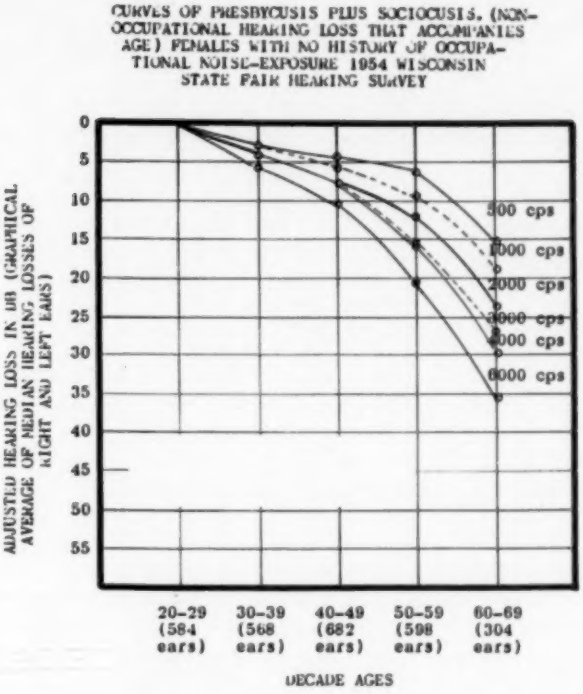


Fig. 6.

Second, in spite of the availability of prefabricated test booths, the environmental conditions under which most industrial audiometry is done leave much to be desired. We are now engaged in an educational campaign to make industrial and military physicians and technicians fully aware of the importance of proper test conditions. The pertinent information is made available in the revised Guide, in journal articles, in special training courses offered for audiometric technicians, etc.

Third, an examination of the methods of recording data reveals variations, inadequacies and errors which make it almost impossible to use much of the available material.

HEARING CONSERVATION DATA CARD NO. □ (1)

A. IDENTIFICATION

Last Name | First | Middle | (2)° Status: Military X□ Civilian Y□ | (2-10)° Social Security No. - Service Number | (10)° Sex: M-Y□ F- X□

B. CURRENT NOISE EXPOSURE

(11-13) Dept. or Location | (14-18) Job or Noise Code (AFSC-MOS) | (19) Time in Job: Mos. 0-11, Years 1-5, 6-10, 11-15, 16-19, 20+ | (20) Exposure Time | (21)° Wears Ear Protection other than Dry Cotton During Exposure to Loud Noise: Always or Frequently 1□; Seldom or Never 2□ | (21)° Most Frequently Used Ear Protection: Class: Insert X□ Covers Y□; Type: Elastic 3□ Non-elastic (waxy) 4□ Rigid (fitted) 5□; Type: Muffs 6□ Helmet 7□ Other 8□

0 X Y 1 2 3 4 5 6 7 8

C. PREVIOUS NOISE EXPOSURE

Time in each Category: (22) a, (23) b, (24) c, (25) d, (26) e, (27) f

(28) Gunfire: Basic training 1□; Combat-Light Arms 2□; Combat-Heavy Arms 3□; Hunting 4□; Target Practice 5□

	(22) a	(23) b	(24) c	(25) d	(26) e	(27) f
Previous Job						
Job before that						
All prior jobs						
Ear protection	X	Y/X	Y/X	Y/X	Y/X	Y/X

D. MEDICAL HISTORY AND STATUS

(29-30): Aural Pain 01; Drainage 02; Ear Injury (mechanical) 03; Surgery (ear or mastoid) 04; Head Injury, with unconsciousness 05; Tinnitus Prior to First Noise Exposure 06; Tinnitus Following Exposure to Noise 07; Hearing Loss in Immediate Family 08

(31-32): Malformation of External Ear or Canal 01; Obstruction of, or Drainage from Canal 02; Perforations of Drumhead 03; Upper Resp. Infect. or Nasal Allergy 04; Tubal Obstruction 05

	A	L
1	□	□
2		
3	□	□
4	□	□
5	□	□
6	□	□
7	□	□
8	□	□

E. MOST RECENT NOISE EXPOSURE

(33) Time Since: Hours 0-23, Days 1-7, 8-14, 15-21, 22-28, Min 29-59, 0-29, 30-59

(34)° Duration of: Hours 1-7, 8-23, Min 0-29, 30-59

(34)° Used Ear Protection: Yes X□ No Y□

	Hours				Min			
	1-7	8-23	1-7	8-14	15-21	22-28	29-59	0-29	30-59
1 2 3 4 5 6 7 8									

Remarks:

F. HEARING LOSSES

(35)° Had Audiometric Test: Before Yes X□ No Y□

(35)° Day of Week | (36-38) Date-No/Yr | (39-40) Age

			Right: Pure Tone							Left: Pure Tone							Speech		
			(41) 250	(42) 500	(43) 1000	(45) 1500	(47) 2000	(49) 3000	(51) 4000	(53) 6000	(55) 8000	(57) 250	(59) 500	(61) 1000	(63) 1500	(65) 2000	(67) 3000	(69) 4000	(71) 6000	(73) Rt.	(75) Lft.	(77) Bin-aural

PREPARED BY THE RESEARCH CENTER OF THE SUBCOMMITTEE ON NOISE IN INDUSTRY, 111 NO. BONNIE BRAE, LOS ANGELES 26, CALIF.

Fig. 7.

Recognizing the importance of accurate records, we devised a standard form for recording hearing conservation data. This form, called a "Hearing Conservation Data Card" is shown in Fig. 7. The Data Card is unquestionably a step in the right direction, but undoubtedly it will be years before it will be accepted by industry in general.

Fourth, one of the worst problems to contend with in the use of industrial data is the rapid turnover of personnel. Very few noisy industries employ the same personnel over long enough periods of time for us to make fruitful studies of hearing loss. The aircraft companies, for example, have almost a 100 per cent turnover per year in their noisy departments. Consequently, it is unusual to be able to follow the same person with repeat tests for more than a few months at a time. For this reason, we must obtain very large amounts of data if we are to draw valid conclusions about industrial hearing loss in general.

Fifth, audiometric data is subject to the usual errors of sampling and measurement. Such errors may make measured values consistently larger than or smaller than the "true" value. Variable errors may cause measured values to fluctuate from one occasion to another and thereby "hide" changes or relationships in a maze of variable unstable numbers. Company policy for assigning men to various jobs may give rise to sampling bias by producing an occupational group within the company that is not typical of the occupation in industry as a whole. Errors may result from failure to control relevant factors while measurements are being made. Audiometric measurements, for example, are known to be influenced by such effects as practice and fatigue. Experimenters must be careful to equate comparison groups in such matters as age, sex, etc. The calibration of the equipment must not be changed between the time of testing one comparison group and another.

CONCLUSIONS.

In spite of the many very real difficulties that are encountered in field studies of industrial hearing loss we feel that industrial and military audiometric data can be used to good advantage if they are properly recorded and if the necessary supplementary information is included in each record. For research purposes, industrial records should contain *at least, 1.* the age, in years, of the person tested; *2.* a standardized specification of the noise environment; *3.* an accurate record of the man's exposure history; *4.* a statement of the reason

for making the audiogram, and *5.* the date and time within the work cycle when the measurement was made.

SUMMARY.

Finally, let me summarize the main points of the paper: *1.* Design in industrial field research is largely restricted to appropriate tailoring of existing data production methods. It is unlike laboratory research where experiments can be designed to yield the type of data needed, and since manipulative controls are not feasible, statistical controls must be provided during analysis of the data—a situation which generally requires the collection of very large amounts of data. *2.* Many restrictions are imposed on the use of audiometric data obtained through usual industrial channels, because most industrial groups are not homogeneous as to noise-exposure history, job continuity, use of ear protection, and time of hearing test during a day. Further, because companies differ in their method of record keeping, the data are likely to vary in respects that may limit the usefulness of the information in studies which combine company samples. Although such restrictions do not make the data useless they do point out the need for careful scrutiny and skillful analysis if valid conclusions are to be drawn. *3.* There are a large number of specific factors which can reduce the validity of data collected in industrial situations. Some of these factors can be eliminated. For example, poor testing environments, poorly trained audiometric technicians, obvious errors in data recording, and nonstandard or inadequate methods of data recording. On the other hand, there are many conditions which cannot be altered. For example, it is improbable that industry in general will be able to make all audiograms after a period of absence from noise-exposure. *4.* Data will be more useful if the forms used to record the data are standardized. In a standard form particular attention should be paid to details of noise-exposure, to time of hearing tests, and to the reason for the hearing tests. *5.* Explanations for some of the discrepancies disclosed in industrial data are (a) biases operating in the selection of the sample and (b) nonrepresentativeness of the audiograms. *6.* In spite of restrictions

and limitations, future industrial data can be used to good advantage if certain remediable faults are corrected.

A LABORATORY METHOD FOR THE STUDY OF ACOUSTIC TRAUMA.*

DONALD H. ELDREDGE,
and
WALTER P. COVELL,

Central Institute for the Deaf and Washington University,

St. Louis, Mo.

To begin I should like to stress two points: first, the work to be discussed here is part of a continuing project to learn more about acoustic injury of the ear. Many others working at the Central Institute for the Deaf, especially Drs. Davis, Riesco-MacClure, Fernández, Tasaki, Legouix, Goldstein and Benson, have made major contributions to these techniques in the past. Dr. Davis continues as our most helpful advisor, and, when he has time left over from his many other duties and researches, is still an active collaborator.

Second, we are really studying acoustic trauma in the laboratory, and this is quite different from the subject matter of the other presentations of this morning. Ideally, in planning this program Drs. Silverman and Hirsh would like to have had someone describe the pathologic physiology of human ears which had been exposed to the noise of, say a drop forge for 5, 10, 20 and 30 years. This is not possible, and so we are to discuss acoustic injuries to the guinea pig ear. We would like to know about real deficits in hearing function, but we must look at cochlear potentials and cochlear anatomy in the guinea pig. We would like to know about exposures that continue for years; instead, our experimental exposures last minutes to hours. Although some results from our experiments will be presented, the title, "A Laboratory Method for

*Supported by Contract No. AF 33(616)-3637 between Washington University and the Bio-Acoustic Branch of the Aero Medical Laboratory, Wright Air Development Center.

Reproduction in whole or in part is permitted for any purpose of the United States Government.

the Study of Acoustic Trauma," was chosen to emphasize that we are not talking about Hearing Loss and Noise Exposure in the main sense of this session.

Some time ago Hallowell Davis[1] and the numerous collaborators mentioned earlier noted a relation between the "threshold" shift for the cochlear microphonic as recorded by differ-

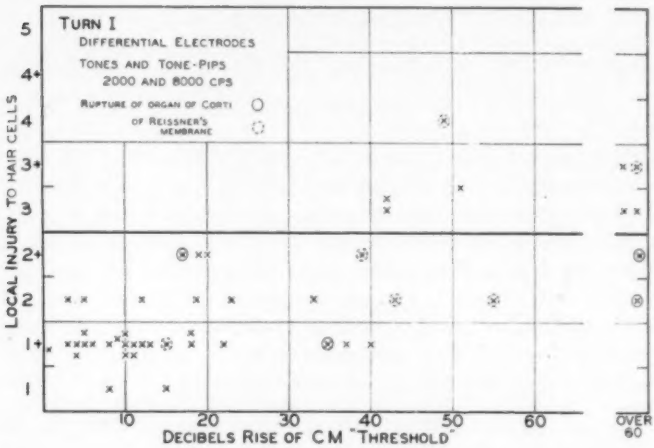


Fig. 1. The elevation of the "threshold" of the cochlear microphonic as measured in Turn I by differential electrodes. The ordinant is an anatomical injury rating scale of nine points. The heavy horizontal line divides the probably permanent injuries to hair cells (above) from the probably reversible injuries (below). The crosses represent differential electrodes.

ential electrodes placed in scala vestibuli and scala tympani, and the anatomical injury observed between these electrodes after acoustic trauma. This relation is shown in Fig. 1. The ordinant is an anatomical injury rating scale of nine points. Injury ratings of 3, 3+, 4, and 4+ were considered to represent probably permanent injury to the hair cells of the organ of Corti, and were associated with "threshold" shifts of the cochlear microphonic potential of about 40-60 decibels. Fig. 2, taken from one of our more recent papers,[2] shows the same relation measured after the ears had been allowed to recover from the acoustic trauma for periods up to 14 days. Although

the values for "threshold" shift corresponding to these injuries is slightly less, the general trend is the same.

The preparation of good temporal bone specimens for microscopic study is a slow tedious process, and the "feedback" of information from one experiment to the next is very slow if we rely solely on this method; however, one knows the "threshold" shift for the CM immediately, and using the information from this slide, can quickly estimate the anatomical degree of an acoustic injury. In addition, other electro-

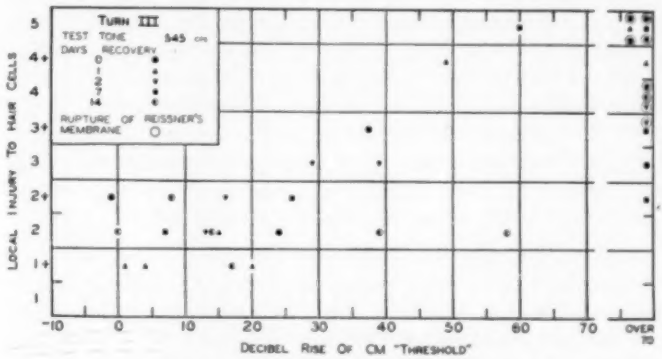


Fig. 2. Local injury to the hair cells in the third turn plotted against decibels rise of CM "threshold" for 500 c.p.s. in the third turn. The symbols correspond to the different durations of the recovery period.

physiological potentials can be measured. When these are compared to each other and to the anatomical injuries later observed, there is a good possibility that our understanding of the pathologic physiology of acoustic trauma will be improved. These ideas were basic to our plans for the experiments I will describe.

Now let us ask a question about acoustic trauma. What reciprocal changes in the intensity and duration of an acoustic stimulus will produce a constant injury to the organ of Corti? For years we have been aware that brief, but intense, stimuli (for example, an explosion) can leave a permanent hearing loss. Other stimuli which are apparently harmless for short

exposures can produce permanent hearing losses if the exposure is made sufficiently long. There is a tremendous continuum between these durations, measured in milliseconds on the one hand and years on the other. The first experiment to be described deals with durations of stimuli only between one and 160 minutes. We consider these durations to fall in the category of acute acoustic trauma, not chronic noise-induced hearing loss.

For the traumatic stimulus we used a 500 c.p.s. tone generated by an enclosed acoustic system sealed to the ear canal of the guinea pig. This acoustic system was very stable, and carefully calibrated so that we could specify the sound pressure level of the stimuli at the tympanic membrane within ±2 db. Remembering yesterday's presentations, a 500 c.p.s. tone will stimulate all the turns of the cochlea nearly to the apex. The greatest amplitude of motion in the basilar membrane will appear in the third turn. We would expect the more severe injuries here also, with lesser or possibly no injuries elsewhere along the basilar membrane.

Pairs of differential electrodes recording from the first and the third turns were used to measure the cochlear microphonics generated at these two locations. The "threshold" shift for the cochlear microphonic produced by a 500 c.p.s. test tone, and measured from the third turn, should indicate the more severe injury. The same measure, "threshold" shift for the CM produced by test tones (we used 500, 2000 and 8000 c.p.s.) recorded from the first turn should indicate the degree of anatomical injury in the first turn. To avoid the effects of transitory fatigue, the measurements were made one hour after the end of the traumatic stimulus.

Taking some clues about critical exposures from previous experiments, we set about exposing guinea pig ears to the 500 c.p.s. tone, planning the experiments as we went along. The idea was to use exposures which would "bracket" our criterion injury as indicated by "threshold" shifts, measured in the third turn for a 500 c.p.s. test tone. In Fig. 3 are shown the exposures used and the average "threshold" shifts for the various exposure conditions. Each point represents one group of exposures to the sound pressure level shown on the ordin-

ant, and the duration plotted logarithmically in minutes on the abscissa. The number of ears in each group varied from one to ten. Five to ten ears were used for exposures which gave 40 to 60 db. "threshold" shifts, whereas only one, two or three were used to explore the exposures above and below this range. Open circles designate average shifts of less

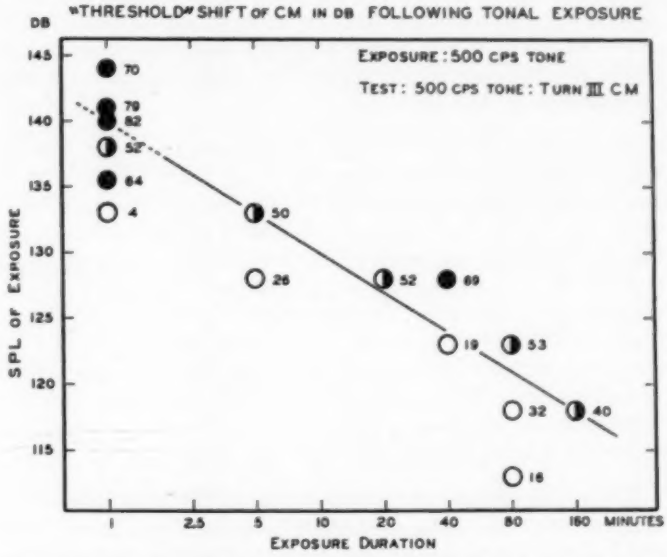


Fig. 3. Sound pressure levels and durations of exposures to 500 c.p.s. tone. Numbers indicate average "threshold" shift for CM from Turn III in response to 500 c.p.s. test tone. Open circles indicate exposures which produced less than 40 db. shift; half-filled circles indicate 40 to 60 db. shift; and solid circles indicate more than 60 db. shift. Line described in text.

than 40 db., and filled circles represent average shifts of more than 60 db. The half-filled circles designate the exposures which produced our criterion shift of 40 to 60 db.

For a moment let us turn to the problem of chronic noise-induced hearing loss. In trying to arrive at some reasonable way to relate the contributions of sound pressure level and duration of exposures to the hazard of noise exposure, an

equal-energy hypothesis has been proposed by Eldred, Gannon and von Gierke.[3] Although there are no direct data to support this hypothesis, the relations so described do not conflict with common experience, and do appear to be more reasonable than other arbitrary assumptions one could make. The hypothesis assumes that noise exposures may be equated with respect to hazard when the total acoustic energies of the exposures are equal. A set of such equal exposures can be described on a double logarithmic plot of sound pressure and duration by a straight line with a slope such that it goes down 3 db. in sound pressure level each time the duration of exposure is doubled.

A line which drops 3 db. for each doubling of duration has been drawn through the exposure point described by 133 db. for five minutes and which gave an average "threshold" shift of 50 db. This line rather well describes the parameter of our 40 to 60 db. "threshold" shift criterion. It does not do so well for the one-minute exposures, and we have reason to suspect that the function described by this parameter is reaching a limit at these sound pressure levels. There must also be a similar lower limit, but this has not been sought systematically. Parenthetically, I should note that this line should not be confused with other equal-energy parameters which have been proposed as safe for human ears.

Before becoming too entranced with the possibilities for speculation on the good agreement between these data and the equal energy hypothesis, we ought to review the pathology produced in these ears. The anatomical injuries to the hair cells of the organ of Corti in the third turn were rated on the same nine-point scale which was the abscissa of the first slides. In Fig. 4 the average injury ratings are depicted by the blackened sectors of the circles. These circles are, of course, located in the same exposure position as on Fig. 3, and the same equal-energy line has also been carried forward to provide a common reference. It is clear that the degree of injury was primarily proportional to sound pressure level, although longer durations of exposure did slightly increase the injury ratings. The injury ratings of some of the peripheral points invite a new line, which might have a slope such

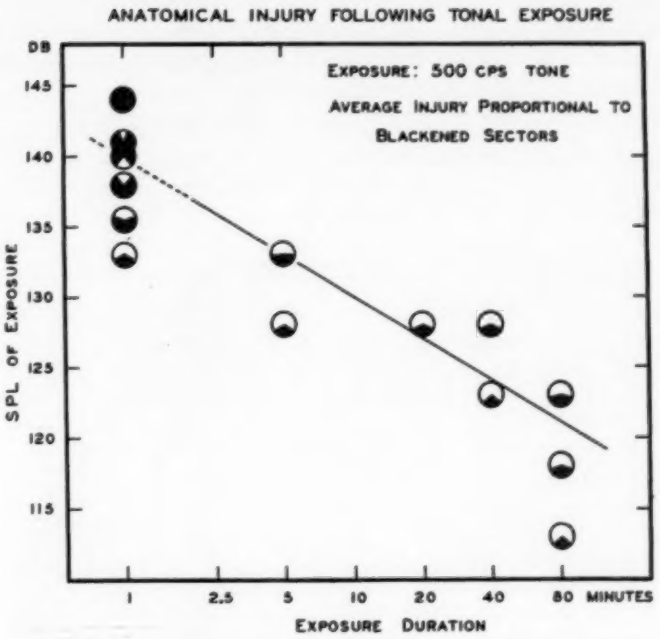


Fig. 4. Sound pressure levels and duration of exposures to 500 c.p.s. tone as in Fig. 3. Injury of hair cells in Turn III proportional to amount of blackening of circles.

that the sound pressure level decreased only one decibel each time the duration of exposure is doubled; however, we must do the experiments which would "bracket" this new parameter before the parameter can be drawn with any degree of assurance. In particular, we must learn the sound pressure thresholds for severe injuries at the longer durations of exposure.

The best we can do so far is to alter our concept of the relation between anatomical injury and "threshold" shift for the cochlear microphonic responses. Both can be produced by acoustic trauma, but each relates differently to the exposure dimensions of pressure and duration. There is the further condition that definite injury to the organ of Corti

nearly always produces a deficit in the cochlear microphonics as we measure them, whereas a deficit in the cochlear microphonic response is not always accompanied by the definite injuries we are accustomed to recognize as such.

In Fig. 5 is shown one injury that is more difficult to interpret in terms of probable function than others we have seen more often in the past. The hair cells of the organ of Corti

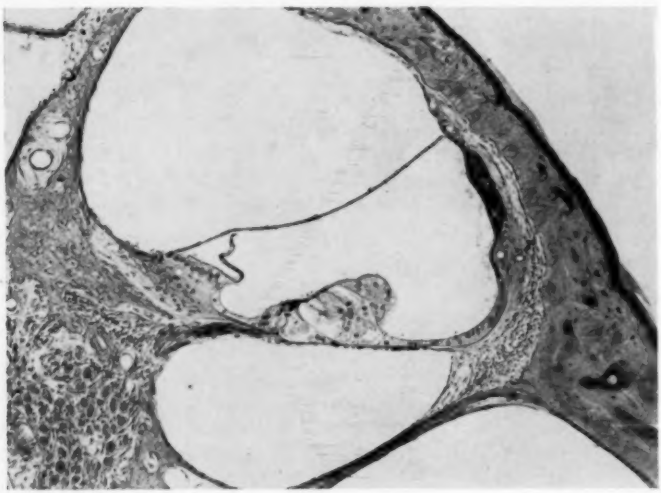


Fig. 5. Photomicrograph of a section through the third turn of a guinea pig cochlea, showing reasonably intact organ of Corti, except for Deiters cells. Some are detached from the basilar membrane and globules of their protoplasm appear in the tunnel of Corti—115X.

do not appear badly injured, but the Deiters cells supporting them have been injured to the extent that portions of these cells are torn off and deposited in the tunnel of Corti. From other studies[2] we infer that these hair cells should regain a normal appearance, but we do not know whether they will be properly attached to the basilar membrane by the Deiters cells so that they can function normally. Similarly we are not sure of the functional significance of loss of mesothelial cells. Absence of mesothelial cells does not preclude normal

or near-normal cochlear microphonics in the senile guinea pig.[4] Other detailed changes in the supporting cells of the organ of Corti are similar to changes observed by Lindsay, Perlman, Fernández and Hemenway[5] following occlusion of the cochlear blood vessels.

It is quite possible that our original injury rating scale is appropriate only to injuries produced by stimuli which produce motions that exceed certain elastic limits. We may need to develop a new scale for rating the injuries produced by the lower sound pressure and longer duration exposures.

Another question we have asked is "how do pure tones compare with noises in their ability to injure the organ of Corti?" The critical band hypothesis, which has been found extremely useful in psychophysical experiments, suggests one possible relation. Given a flat spectrum noise which just masks a pure tone, only a narrow band of frequencies around the pure tone actively contributes to the masking. This critical band of frequencies has the same acoustic energy as the pure tone. The overall pattern of stimulation on the basilar membrane for the tone and for the critical band can be inferred to be the same or very nearly so, and to be equally broad. If injury to the organ of Corti depends upon the energy actually delivered to a particular region of the basilar membrane we would expect a pure tone and its critical band to be equally injurious.

A 300-600 c.p.s. band of noise contains more frequencies than the critical band for 500 c.p.s., and presumably distributes energy to a larger portion of the basilar membrane than does a 500 c.p.s. tone. If the total energy delivered to the basilar membrane is the same for the 300-600 c.p.s. band of noise and for the 500 c.p.s. tone, the area of the basilar membrane giving the greatest response to 500 c.p.s. tones will have a higher energy density when the stimulus is the tone, than it will have when the stimulus is the band of noise. Data supporting the critical band hypothesis in man tell us that the energy concentration in this area should be about 7 db. more for the pure tone. If the injury at this location on the basilar membrane depends upon the energy density, it should be necessary for the overall rms level of the 300-600

c.p.s. band of noise to be seven db. more than the rms level of the pure tone. The noise should then injure a *longer* segment of the basilar membrane, but the most severe local injuries should be the same. We do not know the width of a critical band for the guinea pig. It should not, however, be different in kind if the critical band hypothesis depends on the space-time pattern of the waves in the basilar membrane, as is assumed for the purposes of this experiment.

It is also possible that peak displacements of the basilar membrane, rather than average energy level, are primarily responsible for the injuries we observe. This hypothesis depends on the concept of exceeding some elastic limit in the tissues. The peak displacements produced by a pure tone are, of course, uniform with respect to time. A band of noise of the same overall level will produce displacements of random amplitude both greater and less than that of the pure tone. Considerably greater displacements will occur occasionally and could produce more severe injuries. We have not tried to quantify the differences, because we do not know the elastic limits of these tissues; however, if injury does depend on peak displacement, the overall sound pressure level of a band of noise required to produce a given injury will be less than that of the pure tone.

It is, of course, also possible that the injury may in some way depend primarily on the total energy delivered to the cochlea. In this case the injuries should be the same when the overall rms levels of the band of noise and the pure tone are the same.

Again using the 40 to 60 db. "threshold" shift of the CM, recorded from the third turn, to a 500 c.p.s. test tone as our criterion, we have exposed guinea pig ears to a carefully calibrated 300-600 c.p.s. band of noise. The results are shown in Fig. 6. The coordinants and the symbols are the same as for Fig. 3, showing the "threshold" shifts produced by the pure tones. The line which corresponded to the 40 to 60 db. "threshold" shift parameter for the pure tone exposures also appears for comparison.

For the five-minute exposure the sound pressure level of an

exposure condition which produced a 40 to 60 db. "threshold" shift for the CM from the third turn is six db. below the corresponding sound pressure level for the pure tone. For this exposure condition we must clearly accept the hypothesis that the peak displacements are primarily responsible for the "threshold" shifts observed. At the lower levels and longer

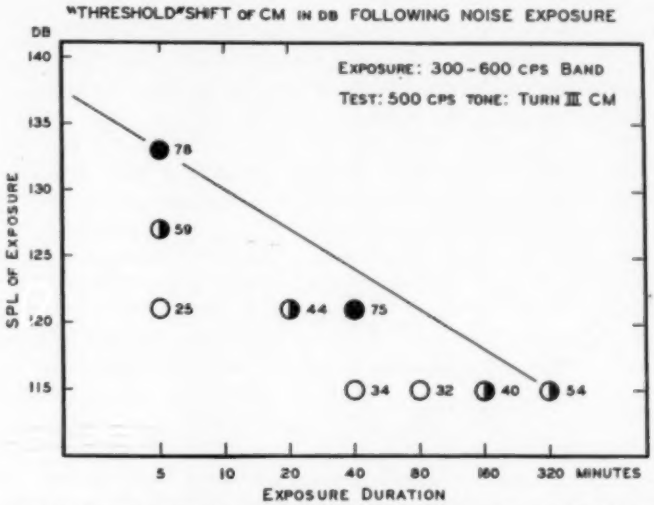


Fig. 6. Sound pressure levels and duration of exposures to 300-600 c.p.s. band of noise. Numbers indicate average "threshold" shift for CM from Turn III in response to 500 c.p.s. test tone. Open circles indicate exposures which produced less than 40 db. shift; and solid circles indicate more than 60 db. shift. Line is from Fig. 3 for comparison.

durations there is a tendency for the exposure conditions yielding the criterion "threshold" shifts to approach this parameter for the pure tone exposures. The data are not sufficient to determine really precise locations or limits for either set of experiments. Rather than drawing a second line for the band of noise experiments, we prefer to state that it is different from that for the pure tone and starts at lower sound pressure levels. Already we are stretching the time limits for acute experiments of this kind. Both the tone and the band of noise parameters could be approaching the same

limiting lower sound pressure level, or it is quite possible that the lines may cross, and at even lower levels and longer durations of exposure they may assume the relation predicted by the critical band hypothesis. Chronic experiments will be necessary before these questions can be resolved.

The temporal bone specimens injured by the band of noise are quite comparable to those injured by the pure tones with respect to the amount of injury to hair cells. As for the exposures to the 500 c.p.s. tone, the shorter, higher-level exposures injured the hair cells more severely than the longer, lower-level exposures. Both stimuli produced maximum degree of injury in the third turn of the cochlea. The areas of injury differed in that the injuries produced by the tone often spread to the upper part of the second turn, while the injuries produced by the band of noise spread toward the fourth turn and often to the apex. It is our impression that there may be other differences. The displacements of cells within the organ of Corti appear relatively greater and more obvious following the noise exposures, and gross rupture of the organ of Corti and of the reticular lamina are less frequent. These displacements are often accompanied by deformation of the tunnel of Corti and by a more frequent swelling of Deiters cells, which then appear to encroach upon and deform the hair cells. The internal phalangeal cell may be tremendously enlarged and the internal hair cell actually displaced. Stretching and rupture of Reissner's membrane occurs more frequently following the longer exposures to the band of noise. For the longest exposures, injury to Reissner's membrane was apparent even when injuries to the hair cells were minor and loss of CM responses only 20 to 30 db. The exposure to the tone (500 c.p.s.), on the other hand, usually appeared to injure more directly the hair cells and the mesothelial cells.

The pathological differences noted are only trends, and may not prove to be significant. Thus we believe the inferences we have drawn from the data on "threshold" shift of the CM responses are correct with respect to the relative hazards of pure tones and bands of noise for the brief intense exposures we have used.

In summary, the "threshold" shift for the cochlear microphonic produced by acute acoustic trauma by a 500 c.p.s. tone obeys the equal-energy hypothesis when the duration of exposure is between five and 160 minutes. A 300-600 c.p.s. band of noise at a lower overall sound pressure level than the 500 c.p.s. tone produces the same threshold shift of the cochlear microphonic as the tone. From this we conclude that the shifts are related to peak pressures of the stimuli rather than to the energy of the stimuli. The same pathology is not observed for all exposure conditions which produce the same threshold shifts of the cochlear microphonic. This relation of the observed pathology is consistent with the finding that peak pressures play such an important role in acute acoustic trauma.

REFERENCES.

1. Davis, H., et al.: Acoustic Trauma in the Guinea Pig. *Jour. Acoust. Soc. Amer.*, 25:1180-1189, 1952.

2. Eldredge, D. H.; Covell, W. P., and Davis, H.: Recovery from Acoustic Trauma in the Guinea Pig. The Laryngoscope, 57:66-84, 1957.

3. Eldred, K. M.; Gannon, W. F., and von Gierke, H.: "Criteria for Short Time Exposure of Personnel to High Intensity Jet Aircraft Noise." U.S.A.F., WADC Technical Note 55-355, Wright Air Development Center, September, 1955.

4. Covell, W. P., and Rogers, J. B.: Pathologic Changes in the Inner Ears of Senile Guinea Pigs. The Laryngoscope, 57:118-129, 1957.

5. Lindsay, J. R.; Perlman, H. B.; Fernandez, C., and Hemenway, W. G.: Histopathology of Sudden Vascular Disorder of the Labyrinth. Exhibit S-2, Sixth International Congress of Otolaryngology, May 5-9, 1957, Washington, D. C.

NOISE-INDUCED HEARING LOSS. DIAGNOSIS, PRESBYCUSIS, SUSCEPTIBILITY.*

Gordon D. Hoople,

State University of New York,

Syracuse, New York.

The diagnosis of noise-induced hearing loss is not a simple problem. It would be convenient, indeed, if noise produced

*Due to Dr. Hoople's illness, this paper was read by Dr. Ben H. Senturia of St. Louis.

a hearing loss, the audiogram of which would differ materially from the audiograms of every other known cause of hearing loss. Since this is not the case other factors in addition to an audiogram must be taken into consideration.

In the differential diagnosis of hearing losses, it is probable that the history given by the patient is one of the most important factors to be appraised. Obviously, if there has been no exposure at all to noise, no matter what the audiometric curve may be, a demonstrated loss cannot be due to noise; however, if there is sufficient exposure to noise *and* hearing loss of the type usually found in patients who have been subjected to noise, then the demonstrated loss is probably due to this cause. How comforting it would be if the last part of the preceding sentence could read, "the demonstrated loss is surely due to noise." It is rare, medically, that one can be as positive as this. This is because, with man and his diseases, the blacks and the whites do not stand apart; they are mixed. Thus, one is confronted with the problem of ascertaining the amount of black and the amount of white in the presenting grey. This problem comes to the fore when patients who have been exposed to noise, and who have a demonstrable loss, are brought before juries or compensation referees, and doctors from one side testify there is little hearing loss due to noise, and medical testimony from the other side expresses the opinion that most of the loss is due to noise. Both sides admit they are talking in terms of grey, but they cannot agree on the percentages of the black and white which make up the grey; thus, it is that judgment must come into this picture when all of the facts of a given case have been brought to light.

With this in mind, there are several aspects in the history of such patients which can lend weight to one decision or the other.

1. The character of the onset of any hearing loss. This symposium is concerned with industrial noise-induced hearing loss. Sudden explosive noises are not included. These sudden noises, if they are sufficiently loud, often produce a sudden hearing loss. With industrial noise, the opposite of

sudden explosive noise, it is conceivable though *highly* improbable that an exceptional susceptibility might account for a permanent sudden hearing loss. This would be most unusual. I have never seen such a case and have never heard of one, and inquiry among my colleagues yields a like experience; hence, there must be a history of gradual loss. Usually the discovery of such a loss is made by the family or friends of the patient. The loss is so gradual (when at last it begins to be a slight handicap) that the patient does not realize its presence.

2. Associated vertigo. Sudden loss may have accompanying vertigo, but there is none with chronic noise-induced loss.

3. Accompanying tinnitus. If this is present it is more than likely that one can elicit a history of an increase of its intensity late in the day of exposure, or in the following early evening hours. It is also likely that a worker will state that his hearing seems to be poorer during these same time periods.

4. There is always some hearing loss in both ears, although one ear may have sustained greater loss than the other. Careful inquiry into the exact nature of the individual's work pattern and his exposure to noise may reveal a cause for greater loss in one ear than the other. In the great majority, however, the loss is essentially the same in both ears.

5. Further inquiry as to the patient's work conditions may suggest reasons why a loss is present. Much noise in small enclosures or rooms with reverberating walls will cause more loss than similar noise in open spaces. Work with pneumatic hammers, pneumatic drills, drop forges, riveting guns and chipping tools are notable for their contributions to hearing loss. If further evidence of work conditions are available such as the overall intensity of the noise, its spectral characteristics, and the length of time an individual has been exposed to these noise conditions, judgment can be more correctly given as to the cause of a worker's hearing loss. The time factor is most important. Without considerable time spent in noise there will be no permanent loss. In truth, there is no such thing as a hazardous noise in industry. There is only hazardous noise *exposure.*

6. It should be obvious that inquiry should be made for other possible causes of hearing loss in a given patient. This inquiry should be directed, not only to the ears but also to any diseases or accidents which might be contributing factors to a hearing loss. This might prove to be time consuming, but the history should go even further to include the hearing experience of all known relatives.

From these points it can be readily seen that the history can contribute much toward a correct decision as to how much black and how much white there may be in a given case, and certainly shows that a careful history should never be omitted in assaying a suspected noise-induced hearing loss.

The second tool in the hand of the diagnostician, and one which is invaluable in arriving at a correct decision, is the examination of the patient as well as the assessment of his hearing. Here, also, there are points of distinct value.

1. For this discussion we will pass quickly over the general examination of the patient, both physical and laboratory, but this may elicit a reason or reasons for a hearing loss.

2. Theoretically, the examination of the ears, nose and throat should be normal in an individual with noise-induced hearing loss for exposure to chronic noise causes no injury to these parts, *i.e.*, there should be a normal drum head; but this is almost tantamount to saying that noise cannot cause hearing loss in an ear which has been the victim of previous disease. While a well marked conductive loss can give some protection, industrial noise can and does cause changes in the cochlea of such ears. In this area are some of the most difficult decisions an otologist has to make, *i.e.*, how much of the loss of hearing that is present is due to noise and how much is due to the concomitant conductive lesion.

3. More substantial aid is given the examiner by hearing tests. Significant findings include:

a. Bone conduction readings which parallel the air curves. Should a bone-air gap be present some additional cause for the loss other than noise must be in the picture.

b. The air curve will show a loss which is greater at 4,000

c.p.s. than any other frequency in almost every instance. This is always true when assaying an early loss. It is probably so in approximately 98 per cent of all cases, even when long exposure is known to be in the picture (see Fig. 1).

c. The frequencies below 500 are rarely affected. If hearing at these frequencies is depressed, search should be made for other causes.

	100	250	500	1000	2000	4000	6000	8000	11000
A.S.	0	–5	–5	–5	–5	40	30	20	....
Air—									
A.D.	0	0	0	0	0	40	35	25	....

Fig. 1.

RIVETER.

	100	250	500	1000	2000	4000	6000	8000	11000
A.S.	5	10	15	55	55	60	50	60	....
Air—									
A.D.	15	15	20	65	65	60	45	55	....

The reading at 4,000 c.p.s. in the Left Ear is the greater.

CHIPPER.

	100	250	500	1000	2000	4000	6000	8000	11000
A.S.	5	10	35	50	50	50	45	60	....
Air—									
A.D.	10	20	40	40	55	60	50	50	....

The reading at 4,000 c.p.s. in the Right Ear is the greater.

MACHINE HAMMER.

	100	250	500	1000	2000	4000	6000	8000	11000
A.S.	–10	–5	0	20	65	85	....	....	....
Air—									
A.D.	–5	0	0	15	70	90	....	....	....

Fig. 2.

d. When there has been lengthy exposure to certain noises and the resultant hearing loss shows a fair amount of change at 1,000 c.p.s., and even at 500 c.p.s. the audiometric dip at 4,000 c.p.s. tends to level out, or even be eradicated, but the audiogram still is indicative of possible noise induced etiology (see Fig. 2).

e. Concomitant disease and presbycusis are two important causes for an eradication of a notching 4,000 c.p.s. audiogram

by causing even greater losses at the 6,000 and 8,000 frequencies. Diseases and toxic conditions which may depress the hearing in this manner are syphilis, dental, tonsillar and prostatic diseases, alcohol, nicotine, quinine, the salicylates, the mycins (antibiotics), senile vascular changes, allergy, etc. (see Fig. 3). (Example: losses above 4,000 c.p.s. are the greatest.)

PRESBYCUSIS.

	100	250	500	1000	2000	4000	6000	8000	11000
A.S.	5	10	10	10	10	20	50	60	....
Air—									
A.D.	0	5	5	5	10	30	60	65	....

DISEASES.

	100	250	500	1000	2000	4000	6000	8000	11000
A.S.	35	40	50	55	60	55	65	70	....
Air—									
A.D.	40	35	45	50	60	65	60	70	....

ALLERGY.

	100	250	500	1000	2000	4000	6000	8000	11000
A.S.	25	25	30	35	50	60	70	75	....
Air—									
A.D.	30	25	35	35	45	60	65	70	....

Fig. 3.

f. Primarily the audiometric findings must be stable. Three audiograms, each taken one week apart and at least 40 hours after the last exposure to noise, must show no appreciable variation. This should be a requirement for establishing a base line for noise-induced hearing loss. This practice, if adopted, rules out any temporary threshold shift of hearing. This temporary threshold shift is known to exist in all involved ears during and shortly after exposure to noise. Likewise, this practice will establish the persistent hearing loss present after 40 hours, from the last exposure. Any change between the persistent hearing loss and a later determined permanent loss will be minimal.

g. Repeated audiograms will be important evidence in ruling out malingering or a false claim that a loss due to disease is due to noise. Sometimes a single audiogram will do this

(see Fig. 4). (Example: unilateral loss claimed to be due to chronic noise exposure).*

PRESBYCUSIS.

Presbycusis has been mentioned above as a complicating factor in interpreting audiograms when noise inducted hearing loss is suspected. Some loss of sensitivity at some frequencies is to be expected as the normal result of the process of ageing. This has been shown in many studies, but those of Bunch,[1] the World's Fair,[2] the San Diego Fair,[3] and the Wisconsin State Fair,[4] are in reasonable agreement as to the hearing loss which is present due to ageing. It is generally agreed that hearing losses due to noise and presbycusis are

	100	250	500	1000	2000	4000	6000	8000	11000
A.S.	0	0	0	5	0	0	5	10	....
Air—									
A.D.	45	50	60	70	60	50	75	80	....

The loss at 4,000 c.p.s. is considerably less than at 500 and 1,000 c.p.s.

Fig. 4.

additive.[5] The Z24-X2 Committee of the American Acoustical Society pointed out that both presbycusis and noise-induced hearing loss are "intra-cochlear deafness." They are indistinguishable microscopically. Both involve the permanent loss of some sensory cells and their nerve fibers. This Committee felt there was no reason to suppose that presbycusis protects the ear from further injury by noise, nor does it make the ear more sensitive to injury.

An allowance for the loss, due to presbycusis, has been suggested for appraising the hearing loss in workers over 50 years of age. For the frequency 2,000 c.p.s., this loss on the average amounts to about 5 db. in the sixth decade, and 10 db. in the seventh. The losses at 4,000 c.p.s. on the average are about 10 db. and 20 db. respectively, but there are large individual differences.[6]

*See Bibliography.

SUSCEPTIBILITY.

That the human ear is susceptible to hearing loss by exposure to chronic noise has been known for years. Fosbroke's[7] article in *Lancet* was written 137 years ago, and this cause of hearing loss was probably recognized before this early date. How susceptible the ear may be, and whether or not some ears are more susceptible than others has been a debated subject for scores of years. The answer to the susceptibility question is relative rather than absolute. Barr[8] commented that of 100 boilermakers examined by him who had worked at this trade at least four years, none had normal hearing. Boilermaking certainly is an occupation which has a hazardous noise exposure, particularly when a worker is subjected to the reverberating sounds inside a boiler. What about less hazardous noise exposures?

Most writers on the subject of noise-induced hearing loss state that some human ears are more vulnerable than others,[9,10] but no one to date has fashioned a statistical table attempting to delineate what percentage of all ears are, for instance, highly susceptible, very susceptible, moderately susceptible, barely susceptible, etc. If one dared prepare such a table it would have to be for one certain well described and accurately measured noise, existing under known conditions, to which would have to be added the known length of exposure. It is obvious that there would have to be thousands of tables to have any value at all, for the variables are multidimensional; even then such tables would be valueless for protection purposes, for they could not delineate a particular individual and indicate that he would be one of the highly susceptibles.

There has been much searching for a reliable test for sensitivity. Everyone acknowledges its desirability, but to date none of the advocated tests have been validated by practical experience. Tests have been devised, based on marked loss of hearing immediately after exposure to loud noise[11] or on the basis of slow recovery from such a temporary threshold shift,[12,13,14,15] but persistent and permanent hearing losses do not necessarily follow a temporary threshold shift. Here

again variables are multidimensional. Like variables are present for the Lawrence and Blanchard[16] aural harmonics test for susceptibility.

Glorig has stated from the examination of many thousands of industrial hearing records there are only a few persons who are highly susceptible to noise. For the present, then, it seems expedient and wise to employ monitoring audiometry in all industrial situations where a noise problem exists, and to separate from their noisy environment those workers who show significant hearing losses on several 40 hours audiograms. This will protect both the employer and the employees, and is a safe procedure to use till further studies and further statistics can show that a valid susceptibility test is or is not possible.

SUMMARY.

The problem of the diagnosis of noise-induced hearing loss is difficult, because losses due to causes other than noise may be present. A detailed history of a patient may furnish valuable information. The examination of the patient, together with a study of his audiograms, can give further light.

Presbycusis is a known complicating factor, and an allowance for its presence should be made in workers over 50 years of age.

Susceptibility to noise is as yet an unsolved problem. No predictive tests have been validated. The answer to susceptibility is relative rather than absolute.

BIBLIOGRAPHY.

*Confirmation of many of the statements in this first section of the paper can be found in the writings of:

BUNCH, C. C.: THE LARYNGOSCOPE, Vol. 47, 1937.

BUNCH, C. C.: *J.A.M.A.*, 118-588, Feb., 1942.

HOUSE, H. P.: *Arch. Indust. Hygiene and Occupational Med.*, 5:124, Jan., 1952.

PERLMAN, H. B.: *Arch. Otol.*, 34:429, Sept., 1941.

SENSENEY, E. T.: THE LARYNGOSCOPE, 57:142-157, 1947.

1. BUNCH, C. C.: Age Variation in Auditory Acuity. *Arch. Otol.*, 9:625-636, 1929.

2. Steinberg, J. C.; Montgomery, H. C., and Gardner, M. B.: Results of the World's Fair Hearing Tests. *Jour. Acoust. Soc. Amer.*, 12:291-301, 1940.

3. Webster, J. C.; Himes, H. W., and Lichtenstein, M.: San Diego County Fair Hearing Survey. *Jour. Acoust. Soc. Amer.*, 22:473-483, 1950.

4. 1954 Wisconsin State Hearing Survey Monograph. *Trans. of Amer. Acad. Ophthal. and Otol.*, 1957.

5. Report of Z24-X2 Committee. *Bulletin Amer. Acoust. Soc.*, 1954.

6. Committee of Eight, Principles of Hearing Loss. *Trans. Amer. Acad. Ophthal. and Otol.*, 60:826-829, Nov.-Dec., 1956.

7. Fosbroke, J.: *Lancet*, 1:645, 1830-31.

8. Barr, T.: *Brit. Med. Jour.*, 2:675, 1890.

9. Gallagher, J. B., and Goodwin, J. E.: *Arch. Indust. Hyg. and Occupa. Med.*, 6:332, July, 1952.

10. Davis, H., et al.: The Laryngoscope, 56:19, Jan., 1946.

11. Davis, H., et al.: *Acta Otolaryngol.*, Suppl. 88, 1950.

12. Wilson, W. H.: *Ann. Otol.*, 59:399-405, 1950; *Arch. Otolaryngol.*, 37:757-767, 1943; 40:52-59, 1944.

13. Jerger, J. F., and Carhart, R. T.: *Jour. Acoust. Soc. Amer.*, 28:611-613, 1956.

14. Wheeler, D. H.: The Laryngoscope, 59:1328-1338, 1949.

15. Christiansen, Erik: *Acta Otolaryngol.*, 46:2, 1956.

16. Lawrence, M., and Blanchard, C.: *Ind. Med., Surg.*, 23:5, 1954.

EAR PROTECTION.*

J. Zwislocki,†

Harvard University,

Cambridge, Mass.

Most probably, even those of you who do not take any active part in solving the noise problems must have noticed that these problems have been discussed, if not exhaustively, at least extensively at many times and at many places. We have a journal entitled "Noise Control," and a "Handbook of Noise Control." Numerous committees and meetings are devoted to noise problems. Possibly some of you might think that we are making too much noise about noise; however, even

*Supported under Contract Nonr-1866(15) with the Offoce of Naval Research, United States Navy (Project NR142-201, Report PNR-211). Reproduction for any purpose of the United States Government is permitted.

†Now at the Office of Research in Special Education and Rehabilitation, Syracuse University, Syracuse, New York.

those who do think so must admit that, one way or the other, we do have too much noise, and that something should be done about it.

I consider myself fortunate to have been given the opportunity of discussing one of the means of combating noise. This gives me the feeling of usefulness, of doing something positive instead of just complaining. Of course I must admit that if nobody complained about noise, and there are still some left who do, I would have no business talking about ear protection.

Now, back to the subject. I doubt that anybody considers the personal ear protectors as the most desirable means of noise control. There are probably no more people who want to wear earplugs or earmuffs than there are people who want to wear eyeglasses. Although not the most desirable, personal ear protectors are probably the most versatile, effective and the most economical means of noise control. This does not imply, of course, that other means should be happily abandoned. In many situations we have to apply every trick we know to keep noise at a tolerable level, and sometimes we run out of tricks. In addition, personal ear protectors are always somewhat uncomfortable and cumbersome, and, maybe most important, they do not improve speech communication by any considerable amount, except when the transmission occurs through an earphone, and the earphone with its socket serves as an ear protector.

The best means of noise control is undoubtedly to stop the noise right at its source. That means, not to make any noise at all. Unfortunately, we have not been very successful in this endeavor and, according to Dr. Knudsen, noise is increasing at a rate of one db. per year. It seems, therefore, that ear protectors are here to stay for a while, and they should be considered at an equal level with the other tools of noise control.

I could devote this time to showing you pictures of various types of ear protectors, but I doubt that in this way you would learn much more than the fact that there are many types of ear protectors. I shall try rather to synthesize our

knowledge on the problem of personal ear protection, discuss the basic requirements, the possibilities, and the main trends in recent design.

It is possible to classify the ear protectors according to their position relative to the ear. We have four kinds: earplugs, semi-inserts, earmuffs, and the helmet. Earplugs are secured in the ear canal and usually do not require any additional means of support (see Fig. 1). Semi-inserts are placed at the entrance to the ear canal and are usually supported by a headband. Earmuffs cover the outer ear like an earphone mounted in its socket or cushion. Helmets cover the whole head, or at least most of it.

I am going to omit the semi-inserts, because there are none on the market, and the helmets, because I feel that at present they do not offer any acoustical advantage.

Now, what is expected of ear protectors? *First*, they should attenuate acoustic noise to a safe level, whatever that level is. *Second*, they should be comfortable, or, let us be more realistic and say, they should not be too uncomfortable. *Third*, they should not have any toxic effect on the skin. There are further requirements that are more or less compelling, depending on the situation. They concern the interference with speech communication, the ease with which the ear protectors can be handled, the sanitary aspect and the economic aspect. Some of these requirements are in conflict with each other, and only compromise solutions appear possible. The best known incompatibility is between sound attenuation and comfort. Another one is between attenuation and speech intelligibility, although this one is more apparent than real; but let us start with the first requirement, *i.e.*, sound attenuation.

How much sound *should* be attenuated?

This is certainly a pertinent question. Unfortunately, I cannot answer it, at least, not at this time, because there are probably as many optimal attenuations as there are situations in which the use of ear protectors appears advisable and maybe even more, maybe almost as many as there are people

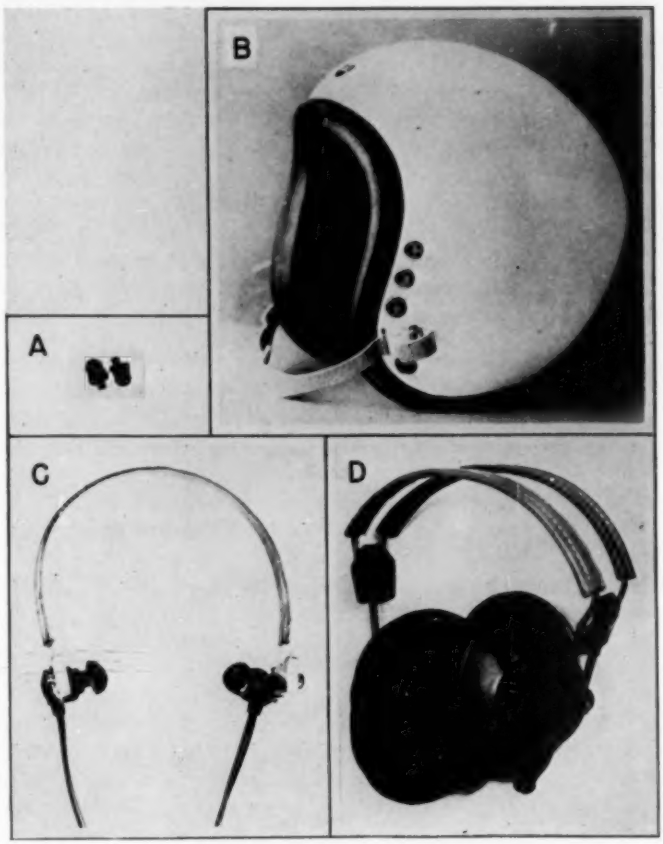


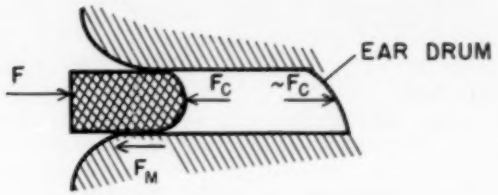
Fig. 1. Four Kinds of Ear Protectors.

involved. In other words, this is a policy question at present, and not a scientific question, and therefore not for me to answer. I can answer, however, a somewhat related question: how much sound *can* be attenuated? This question is restricted, of course, to ear protectors that can be worn either in the ear canals or on the head. By enclosing a man in a cell of concrete, higher sound attenuations than those I am going to speak of can be achieved.

When an earplug is inserted into the ear canal, the sound can still reach the inner ear on three pathways. Because of the flexibility of the skin lining the external auditory meatus, sound waves can produce a vibration of the earplug as a whole. The vibration is controlled at low frequencies by the stiffness and the viscosity of the skin and, at high frequencies, by the inertia of the earplug. The sound can also penetrate the ear through deformation of the earplug—here the stiffness, density and viscosity of the material become important. Finally, sound can reach the inner ear by traveling around the earplug, either via bone or via soft tissue or both. I am going to refer to this channel as body conduction.

For low frequencies, the forces acting on an earplug secured in the ear canal may be schematized as shown in Fig. 2. The force F symbolizes the external sound pressure multiplied by the effective area of the earplug. The force F_C is equal to the produce of the sound pressure generated in the ear canal and of the crosssectional area of the ear canal. Finally, F_M denotes the resultant opposing force developed in the skin lining. The law of the equilibrium of forces requires that F be equal to the sum of F_C and F_M. It is easy to see that the sound attenuation grows with the ratio between F_M and F_C. This ratio is determined to a large extent by the properties of the skin lining and by the volume of the enclosed air, in addition to the acoustic impedance that can be measured at the ear drum. It is, therefore, an anatomical constant and cannot be altered to any considerable degree. The only thing we can do in order to improve the sound attenuation is to make the earplug itself impervious to sound. Various solutions are possible, and I am going to discuss them a little later when I show you some typical designs.

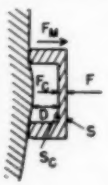
Let us now consider the factors determining the sound attenuation provided by earmuffs. Although earmuffs look considerably different from earplugs, their acoustic action is similar. The vibration of a good earmuff is controlled by the flexibility and the resistance of the skin under the cushion, and by the inertia of the earmuff. The sound attenuation depends mainly on three factors: the mechanical properties of the skin, the mass of the earmuff, and the depth of the



$$F = F_M + F_C \;;\; \frac{F}{F_C} = \frac{F_M}{F_C} + 1$$

$$\frac{F}{F_C} = \frac{F_M + F_C}{F_C} \;;\; A = \frac{p}{p_C} \cong \frac{F}{F_C} \cong \frac{F_M}{F_C}$$

Fig. 2. Schematic drawing of an earplug in the ear canal, and the relations among the acoustic forces acting on the earplug.



$F = F_M + F_C$ $\qquad$ $F = Sp$

$F_M = Z_M u$ $\qquad$ $F_C = S_C p_C$

$F_C = Z_C u$ $\qquad$ $p_C / p = S / S_C \times Z_C / (Z_M + Z_C)$

$F = u(Z_M + Z_C)$ $\qquad$ $Z_C \sim S_C^2 / V \;;\; V = S_C D$

$F_C / F = Z_C / (Z_M + Z_C)$ $\qquad$ $Z_C \sim S_C / D$

$$A = p / p_C \sim Z_M D / S$$

Fig. 3. Schematic drawing of an earmuff covering the ear, and the relations among the acoustic forces acting on the earmuff.

earmuff. The situation is described by the schematic drawing and the formulas of Fig. 3; however, I do not want to go into algebra, and shall state only that, when a high attenuation is desired, the earmuff should be deep and cover as small an area of the head as possible. Of course, in practice there are limits in both respects. A deep earmuff becomes cumbersome, and an earmuff that covers too small an area of the head may exert painful pressure on the pinna and become uncomfortable.

Even if the earplug and the earmuff were ideal in the sense that no sound would be transmitted through them, the sound could still reach the inner ear by body conduction, as I mentioned before. Body conduction constitutes the absolute limit of sound attenuation that can be expected from ear protectors.

The body conduction threshold in a free field has been measured recently, and I am going to report on it in the

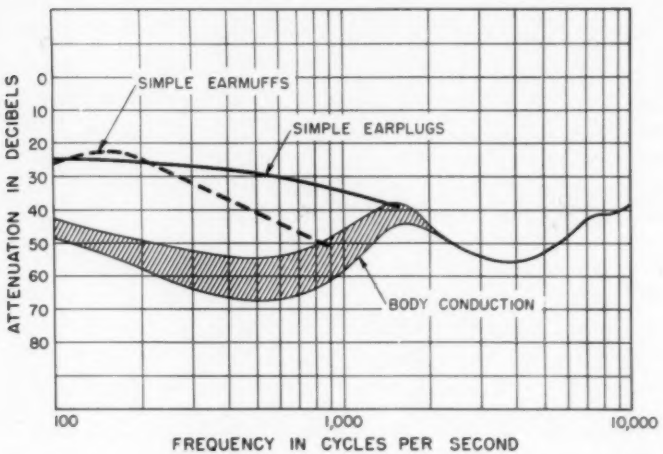


Fig. 4. Air conduction (lower curve) and body conduction thresholds of audibility in a free sound field. The shaded area indicates the uncertainty due to the effect of the occlusion of the ear canal on bone conduction.

coming meeting of the Acoustical Society in New York. Here I can only show you some of the experimental results.

The zero level in Fig. 4 refers to air conduction threshold in a free sound field. The lowest curve in the diagram is the body conduction threshold in a free field as measured recently. The shaded area indicates the uncertainty caused by the effect of the occlusion of the ear on bone conduction. It is well known that the occlusion of the ear canal improves bone conduction at low and medium frequencies when tests are per-

formed with a vibrator; but it is not certain that the same thing happens in a free sound field. The heavy continuous curve shows the expected limit of attenuation for simple earplugs calculated along the lines previously mentioned. The other curve shows the maximum attenuation that can be expected from a simple earmuff. I am stressing the qualification "simple," because substantially greater sound attenuations can be obtained by applying certain acoustic tricks.

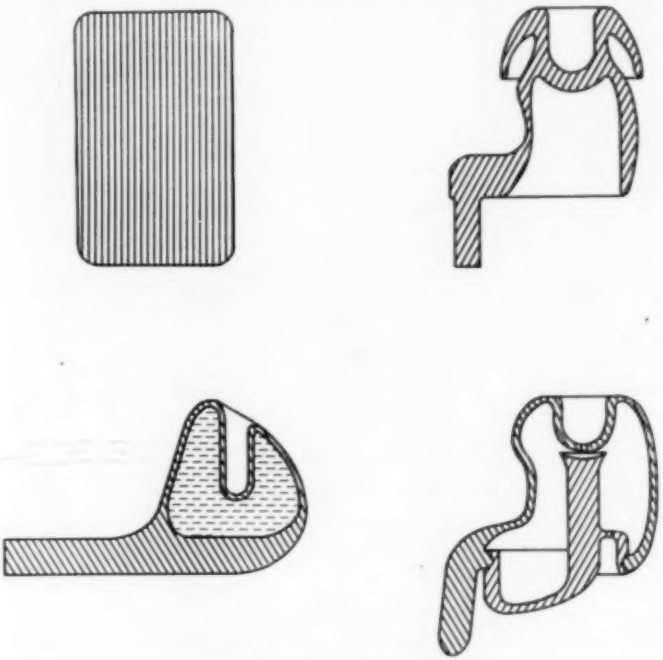


Fig. 5. Four Types of Earplugs.

Regarding a few models of commercial ear protectors, Fig. 5 shows four earplugs which, I think, are typical. The earplug in the upper left corner is a cylinder of wax and cotton. It can be shaped by hand to fit the ear canal. Ear-

plugs of this type are very comfortable and attenuate noise pretty well. They have the drawback, however, that they cannot be washed and, consequently, have to be thrown away after being used only once. Their sticky surface has the tendency to accumulate dirt.

The earplug in the upper right corner consists of rubber or plastic. Because of its elasticity it adapts to the shape of

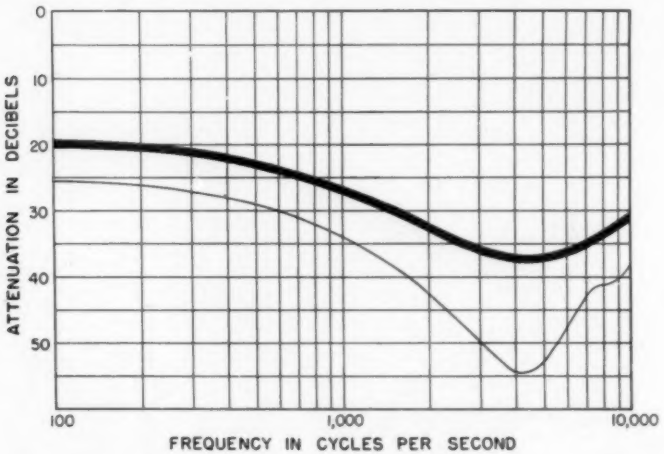


Fig. 6. An approximate attenuation characteristic of the four earplugs shown in Fig. 5 (heavy line). The thin line shows the theoretical upper attenuation limit for simple earplugs.

the ear canal while being inserted. It attenuates noise very well, is washable and can be reused many times. There is one difficulty: in order to obtain a high attenuation, the material of the earplug must be sufficiently stiff. When the earplug is too stiff, however, it becomes uncomfortable, so that only a compromise solution appears possible.

The earplug shown in the lower right corner of Fig. 5 is basically similar to the earplug discussed in the preceding paragraph, but it has some additional features. It contains an internal core made of a stiffer material than the outer shell. This core touches the front wall while the earplug is

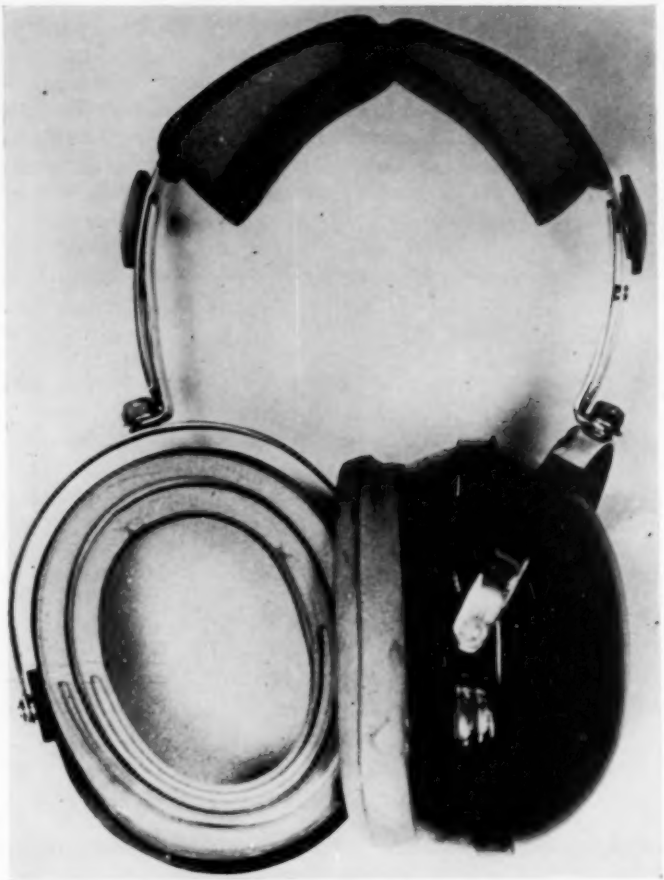


Fig. 7. A Set of Typical Earmuffs.

being inserted into the ear, and facilitates the insertion by stiffening the earplug along its longitudinal axis. In addition, a back wall increases the sound attenuation at high frequencies. In some of the models the front and the back wall are perforated so that the earplug acts as a low-pass filter. The advantages of such a decreased attenuation at low frequencies is still debated.

The earplug in the lower left corner is probably of the most recent design. It combines some features of the wax-cotton earplugs with those of the permanent elastic earplugs. It consists of a malleable core covered by thin elastic walls. The internal tube, which is open at one end, increases the adaptability to the ear canal. Within certain limits the earplug is malleable like the wax-cotton earplugs. At the same time

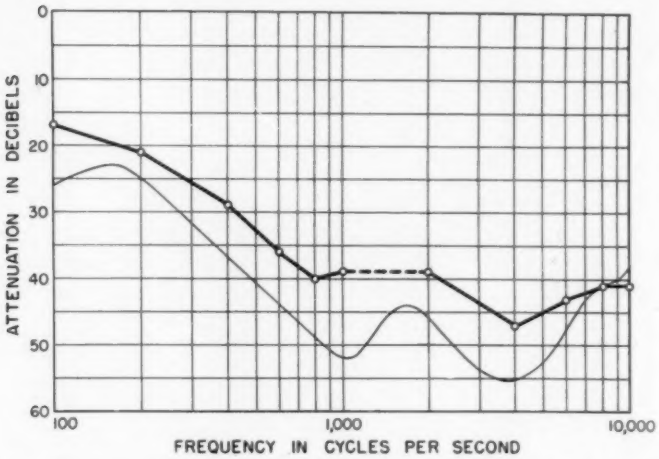


Fig. 8. The attenuation characteristic of the earmuffs of Fig. 7 (heavy line), and the theoretical upper attenuation limit for simple earmuffs (thin line).

it has sufficient resiliency to follow temporal changes of the cross-sectional area of the ear canal. It can be washed and reused.

All the earplugs of Fig. 5 provide approximately the same sound attenuation. At least, the differences among the earplugs, found in one laboratory, are of the same order of magnitude as differences in results obtained for the same earplug in different laboratories. It seems reasonable, therefore, to pool the data obtained for the four earplugs. The thick curve of Fig. 6 was obtained in this way. The thin

curve shows the theoretical upper attenuation limit for simple earplugs.

Fig. 7 shows a set of typical earmuffs. The earmuffs are designed very much along the lines mentioned above: they are deep, enclose a large volume of air, cover a rather small area of the head, and consist of a hard shell and a sealing cushion of foam rubber. The sound attenuation thus provided can be seen in Fig. 8 (heavy line), together with the theoretical limit (thin line). The foam rubber cushion may be considered a weak point in the design of the earmuff. Recently developed cushions with high internal viscosity or with low volume compressibility could further improve the attenuation at low frequencies.

I would like to conclude on an optimistic note by predicting that within a few years we are going to have ear protectors which will attenuate sound up to the limit set by body conduction. I hope that such ear protectors will be commercially available.

REFERENCE.

ZWISLOCKI, J.: "Ear Protectors" in Cyril Harris (Ed.), Handbook of Noise Control. McGraw-Hill, New York, 1957.

ESTABLISHMENT OF CRITERIA BASED ON THE CONCEPT OF NOISE EXPOSURE.*

WALTER A. ROSENBLITH,

Massachusetts Institute of Technology,

Cambridge, Massachusetts.

This morning we have heard a discussion of the general topic of noise-induced hearing loss. Thus far the emphasis

*This work was supported in part by the U. S. Army (Signal Corps), the U. S. Air Force (Office of Scientific Research, Air Research and Development Command), and the U. S. Navy (Office of Naval Research).

has been upon the technical problems that still remain to be solved. The speakers who preceded me stressed for our benefit the difficulties that are encountered in the collection of data and the uncertainties of interpretation that need to be resolved. By way of contrast (and not with any intention of being less honest) I shall try to emphasize, in rather general terms, the knowledge that we have already gained. Consensus is obviously needed before we are ready to go about setting criteria or standards realistically. Unless there is significant agreement among the presumably best informed technical people we have no basis for the social standard of judgment that a criterion represents. In the United States alone, several criteria have been suggested within the past decade, and the same seems to have happened in other countries. This plethora of suggested criteria reflects to some extent the complexity of the task involved, whether we attempt to set standards for maximum allowable concentrations of chemical poisons, for exposure to radiation, or for exposures to noise.

Recently (1956) the U. S. National Academy of Sciences published a series of summary reports on "The Biological Effects of Atomic Radiation." The distinguished committee on the Genetic Effects of Atomic Radiation tried to put before the general reader some of the complications that must be faced before decisions can be reached or recommendations for the general welfare made. The scientist-citizen has to be willing to make rough estimates in areas in which his knowledge is far from certain. He may then have to weigh the outcome of these technical considerations, with their attendant uncertainties, against considerations of national security and international relations. The role of the scientific statesman is thus hardly one for which graduate or even postgraduate study prepares.

THE RELATIONS OF HEARING LOSS TO NOISE EXPOSURE INVOLVE MANY VARIABLES.

Fortunately, the risks that concern us in this conference are less serious than those that result from exposure to atomic radiation; furthermore, we can more easily assess some of

the effects of noise upon exposed humans than we can predict the genetic effects of atomic radiation upon future generations. Just because we can, under the right conditions, make fairly reliable audiometric measurements of man's hearing does not mean that we can ignore the basically multivariate and statistical nature of the after-effects of any exposure, whether this exposure be to acoustic, electromagnetic, or atomic radiation. There are no convenient single numbers that summarize both the exposure and its over-all effects, and this is, to me, what Dr. Hoople means when he writes "the relations of hearing loss to noise exposure are multi-dimensional."

THE SEARCH FOR AN ECONOMICAL DESCRIPTION OF THESE RELATIONS.

It appears, then, that we must pick and choose a set of variables in order to describe noise exposure and its effects upon the hearing of even quite well-defined exposed populations. Selection of the variables is determined in part by our ability to measure certain quantities more accurately and more easily than others, in part by our value judgment of what aspects of human hearing are most important, and finally, by our desire to obtain simple relations between reasonably economical descriptions of both exposures and after-effects. If these relations between quasi-engineering variables make sense in the light of what we know about possible mechanisms, so much the better.

It is in this context that the progress of research on the after-effects of noise exposure and of criterion guessers must be evaluated. We have come a long way since the period in which noise exposure was characterized by an over-all level, read on one of the early sound-level meters; we are, nevertheless, far from being able to give an adequate description of the acoustic environment in which a drop forger spends his working life. We have become keenly aware of the fact that exposures to even quite steady-sounding noises need to be characterized by spectrum, duration, and by what might be called a statistical description of temporal fine-structure.

These considerations give us a glimpse at the complexities

of defining the effective exposure stimulus. Its specification depends, at least in part, upon the kind of after-effect in which we are going to be interested; hence, we need agreement upon a valid and yet reliably measurable estimate of man's auditory capacities. Given our general concern with human communication, it seems reasonable to settle upon man's ability to communicate by speech. This is not the place to discuss our present ability to make such measurements in a nonlaboratory environment, or even the possibility of predicting, by pure-tone audiometry, man's ability to discriminat speech, but we should emphasize that pure-tone audiometry will continue to have value from the viewpoint of preventive medicine for a long time to come.

Finally, we must face the problem of just how homogeneous our populations are with respect to exposure and to the variety of other variables (such as military service, education, and so on) that are at least partially correlated with the response variables that we have chosen.

The socially necessary task of predicting the effects of exposure upon whole populations bears little resemblance to prediction in the classical stimulus-response paradigm. There the stimulus is something that happens "here and now"; it can be assumed that the experimenter has been able to uncouple his subjects sufficiently from their distant and recent past and that post-experimental events will have little influence upon the subject's response behavior. Even in classical psychophysics these assumptions are not always justified; here, they are completely unrealistic. Here, noise exposure stretches over a man's working life-time and may well include significant contributions to this exposure from his military service, from his acoustic environment while he travels to his job or about this globe, and from his addiction to loud music—to name just a few of the possible hazards. These uncertainties in the specification of the exposure make for increased variability, just as presbycusis and quasi-social factors do.

WHOM SHOULD CRITERIA BE DESIGNED TO PROTECT?

In the section entitled "Conclusions" the Z24-X2 report formulated three questions. The committee felt that answers

to these questions or at least decisions regarding them would have to be forthcoming before standards or criteria could be stated. In the years that have passed, informed scientific opinion has come to feel that as long as man's hearing for everyday speech, under everyday conditions, remains protected, we are successfully preventing noise-induced hearing losses from becoming a serious handicap. We have said enough about the difficulties of specifying noise exposure. The last two decades have seen much progress in the spectral analysis of noise; it is to be hoped that in the years ahead comparable progress will be made in the analysis of noise exposures. Certain promising beginnings have already been made, and the necessary electronic equipment and computational facilities are at least in principle available.

The Z24-X2 report asked: "What percentage of the people exposed . . . should a standard be designed to protect?" This amounts to picking a cut-off percentage in a statistically determined situation and is equivalent to saying that a given criterion entails a risk of a serious handicap in X per cent of the exposed population. The representative of a state government who testified before the Z24-X2 committee reminded us gently that such a position was politically impossible in a democracy, where one has to aim at protecting everybody. Without entering into an argument on issues of social philosophy, let us point to the role of programs of hearing conservation and in particular to monitoring audiometry. It is by such programs that the highly susceptible individuals who are working in noise-hazardous areas can be identified and transferred to locations where there is less risk to their hearing. Thus criteria and preventive medicine can combine to protect practically everyone.

Two Examples of Criteria for Noise Exposure.

From what precedes it should be clear that a particular criterion represents a multifaceted compromise: it assumes a certain population and a certain level of acceptable risk; it is, in general, accompanied by a certain hearing conservation program; and it makes certain assumptions about future noise exposures (most often it assumes that they will be more or less identical with those that prevail today). By

making all of these assumptions as explicit as we can, we cut the number of variables to the point where criteria can be set merely by specifying a few numbers.*

Now I should like to quote from two recent documents to illustrate some of the points that have been touched upon: in October, 1956, there appeared an Air Force Regulation entitled "Hazardous Noise Exposure." Its purpose was to establish a program for minimizing the undesirable effects of exposure to noise on Air Force Personnel. In the section of the Regulation that deals with methods of controlling exposure to hazardous noise, "limits for noise exposure" are stated in this manner:

"*a. Limits for lifetime exposure.* The limits for lifetime exposure to broad band noise are given in terms of the band pressure level for four octave bands. The four octave bands are 300-600 c.p.s., 600-1200 c.p.s., 1200-2400 c.p.s., and 2400-4800 c.p.s. In setting limits, it is assumed that the ears are unprotected and exposed continuously during normal workhours over a period of 25 years. The risk of damage to hearing is slight when the pressure level of a band does not exceed 85 db. It increases as the band pressure level increases, and is excessive at 95 db. Ear protection is recommended for continuous exposure to a sound pressure level above 85 db. in any of the four octave bands. It is mandatory for exposure above 95 db. Ear protection may consist of reducing the noise level in the ear canal, reducing exposure time, or both."

There follows a statement on daily short-term exposures in which the so-called equal energy assumption† serves as a

*As a matter of fact, as long as the spectral and temporal character of noise stays in sufficiently narrow bounds, two different exposures of equal duration could be compared on the basis of a single number only. This single number (which could be the over-all level) carries along with it a whole host of hidden parameters.

†According to this concept "equal quantities of acoustic energy entering the ear canal are equally injurious regardless of how they are distributed in time, assuming that the equivalent quantities of noise energy equal or exceed the hearing damage risk criteria." This is not the place to examine the evidence for this assumption or to discuss the ways in which it should be qualified. At present, it represents, within certain limits, a simple and yet quite conservative guide-line. It would, furthermore, be instructive to compare and contrast the rationale for this assumption with the rationales expressed in statements from the NAS-NRC summary reports, according to which "the total dose of radiation is what counts, this statement being based on the fact that the genetic damage done by radiation is cumulative" and "The concept of a safe rate of radiation simply does not make sense if one is concerned with genetic damage to future generations."

basis for a nomogram that permits the computation of equivalent exposure times.

Let us now pass from this example (in which we deal with a rather well-defined population, in which we are preoccupied primarily with "jet-type" noise, and in which the statement of criteria is accompanied by a well-designed hearing conservation program) to a document that aims at less clearly defined situations. In its recent "Guide for Conservation of Hearing in Noise" the Subcommittee on Noise in Industry states:

"At the present time our knowledge of the relations of noise-exposure to hearing loss is much too limited for us to propose safe amounts of noise-exposure. We can, however, point to certain noise levels that indicate when it is advisable to initiate hearing conservation programs. These levels will not be general because a different level is needed for different types of noise and different schedules of exposure. The hearing conservation level that we now specify tentatively applies only to years of exposure to broadband steady noises with relatively flat spectra. It does not apply to short exposures, and, above all, it does not apply to impact noises or narrow-band noises. This tentative hearing conservation level is stated as follows:

"If the sound energy of the noise is distributed more or less evenly throughout the eight octave bands, and if a person is to be exposed to this noise regularly for many hours a day, five days a week, for many years, then: if the noise level in either the 300-600 c.p.s. band or the 600-1200 c.ps. band is 85 db. the initiation of noise-exposure control and tests of hearing is advisable. The more the octave-band levels exceed 85 db., the more urgent is the need for hearing conservation."

These two statements are not unlike in philosophy and general approach, and they do not differ much in terms of the actual numbers that are involved; but it would be unwise to conclude that 85 db. is "it," and that there is not room for different compromises, for different cuts of the criterion-pie. If your situation permits you to "fix" other aspects of noise exposure you may want to state your criterion in minutes per

day, or as a circle of yellow paint around a particular noise source, or even as a single number on the dial of a noise-level meter that weighs the frequency spectrum in an appropriate way.

In summary, I should like to emphasize that, at present, we base our criteria on the concept of *noise exposure* in contradistinction to the previously emphasized *noise level.* We feel that criteria or standards are, in the nature of things, a "package-deal": a given level of risk for a population whose age-structure, health conditions, and "working lifetime expectancy" in a given noise environment are more or less known, is coupled to hearing conservation and noise control programs of specified scope. This ensemble of somewhat uncertain conditions is then held against the Z24-X2 trend curves (or similar information), the facts of speech communication, and against the backdrop of a prevailing social philosophy. This is the process that yields criteria. Men of science often feel ambivalent about their role in this process: more than their nonscientific fellow-citizens, they are aware of the incompleteness of the technical information available; they know that by combining criteria with conservation programs we may protect human hearing at the cost of studying the after-effects of noise exposure more carefully; yet, if those who have expert knowledge will not advise in policy-making, they will fail to make their contribution to the functioning of a democratic society.

BIBLIOGRAPHY.

1. The Biological Effects of Atomic Radiation (Summary Reports from a Study by the National Academy of Sciences) NAS-NRC, Washington, 1956; pp. 108 + XIII.

2. KRYTER, K. D.: The Effects of Noise on Man: *Jour. Speech and Hearing Disorders Monographs*, 1:1-95, 1950.

3. The Relations of Hearing Loss to Noise Exposure (Report by Exploratory Subcommittee Z24-X2). Amer. Stand. Assoc., pp. 1-64, New York, 1954.

4. ROSENBLITH, W. A.; STEVENS, K. N., and the Staff of Bolt, Beranek and Newman, Inc.: Handbook of Acoustic Noise Control, Vol. II: Noise and Man. (WADC Tech. Rep. 52-204 USAF, 1953), pp. 1-262; also available from Office of Tech. Services (Washington 25, D. C.), No. PB 111 274.

5. ROSENBLITH, W. A.: Effects of Noise on Man: Problems for Study. *Noise Control*, Vol. 1, 4:22-27, July, 1955.

6. Principles for Evaluating Hearing Loss (a report of the Council on Physicial Medicine). *J.A.M.A.*, 157:1408-1409, 1955; also *Noise Control*, Vol. 2:11-13, Jan., 1956.

7. Air Force Regulations 160-3 on Hazardous Noise Exposure, Dept. of the Air Force—Washington, Oct. 29, 1956.

8. Guide for Conservation of Hearing in Noise (prepared by the Subcommittee on Noise in Industry of the Amer. Acad. of Ophthalmology and Otolaryngology), pp. 1-24, 1957; also *Noise Control*, pp. 23-31, May, 1957; also *Ind. Med. and Surgery*, Vol. 26:185-192, 1957.

PROF. ROSENBLITH: We will now have the discussion. If you have a discussion as to a specific participant in this morning's program, direct it to him; if not, I shall try to direct it to the person most appropriate.

DR. WARREN H. GARDNER, (Cleveland, Ohio): I have been interested in the statement about the 4000 frequency dip and the probability that the dip can relate to acoustic trauma. I am familiar with the audiograms of perhaps 200,000 to 300,000 children, and I have found quite a large number of 4000 frequency dips in children from ten to 18 years of age. I should like to raise the question: In an adult of 35 years of age, who has been working in a noisy place; how can we tell whether that 4000 frequency is due to acoustic trauma or whether he had that dip when he was 12 years old.

PROF. ROSENBLITH: Will someone volunteer to answer?

DR. SENTURIA: I think Dr. Hoople said something very wise about this precise problem in his paper.

PROF. ROSENBLITH: Would you like to repeat them, or express your opinion?

DR. SENTURIA: No; I would like to express the opinion of Dr. Gardner, that there are many persons under the age of 21—those who have not been exposed to noise, and who show 4000 cycle dips, and I, too, would like a further elaboration of this problem. I wish that Dr. Hoople were here, but since he is not, I would ask someone else in the audience, perhaps Dr. Glorig, to elaborate.

DR. GLORIG: I happen to be one of those persons who do not believe that there is a characteristic noise-induced audiogram. It just so happens that when you look at audiograms of persons exposed to noise, the loss occurs first at 4000 cycles,

but I do not think that because there is a dip there, you can say it is noise-induced. I think the answer to your question is, does it exist prior to it, whether you get an initial audiogram and then have an audiogram to follow, which will indicate what was there before the noise exposure.

PROF. ROSENBLITH: The only time this morning that somebody talked about acoustic trauma was in connection with short exposures that give rise to something that we feel is more adequately described as trauma. The slow, insidious, noise-induced hearing loss that we usually talk about, we would not like to confuse, at least in our present state of ignorance, with what is known as acoustic trauma. Now, this is a preference, this terminology, but it helps us to keep our very limited number of facts straight.

DR. RICHARD E. MARCUS, (Winnetka, Ill.): In discussing the question of differential diagnosis, as raised in Dr. Hoople's paper, and brought up again in the question of the 4096 dip, I wonder whether we might not consider some of these hearing conservation programs and screening surveys. In essence we are finding the number of persons who have a hearing loss. Then, we must use them as a point of departure for the individual hearing test, including the pure-tone audiometry, plus recruitment tests, and whatever other test we may be able to devise, to make the differential diagnosis. The contour of the audiogram itself is not an indication of the diagnosis, but other hearing tests may well give us the clues that we are looking for.

DR. GLORIG: I believe, that if you are going to make a differential diagnosis, you must have more than hearing tests. You need physical examinations and history, particularly history of occupation. I find that one of the facts most lacking in the histories that are taken in noise-induced cases is the history of the occupation of the individual. This, then, allows for not having sufficient noise exposure, or no knowledge of what kind of noise exposure. The making of a differential diagnosis is a difficult procedure and needs a great deal of experienced judgment on the part of the diagnostician. It could not be done at the flip of a finger and

as just a guess, such as I have heard many times in court procedure, where the man was exposed to noise for five minutes. "This is capable of producing a hearing loss; therefore, this man's hearing loss is due to noise." It is a great mistake to do this sort of thing. You get yourself backed up in a corner many times, and can be made a fool of by the attorneys in this respect. I don't think test of recruitment will give you any final answer.

PROF. ROSENBLITH: Not being a medical person, may I say something about the way we have approached this problem. We have talked here this morning about proability tables of hearing loss for given noise exposures. Just as in mortality tables, there will be people who die early and people who die later, so for a given noise exposure, there will be a wide spread, and in this particular thing, you cannot, in my opinion, predict, unless you have a lot of extra knowledge. Just as a hearing conservation program is also part of a diagnosis, because, in order to make a diagnosis, you want to have the kind of information that comes from controlling a man's hearing under conditions of noise exposure.

CONTRIBUTED PAPERS.

SESSION A.

May 14, 1957.

SOME SIMILARITIES BETWEEN HEARING AND SEEING.*

S. S. STEVENS,

Harvard University,

Cambridge, Mass.

One of the most vital and interesting facts about human beings is that they are equipped with sense organs through which they keep in contact with the world around them. We perceive our environment through our sensory receptors, especially through our eyes and ears, and it is difficult to conceive how the human race could survive if we had no eyes or ears to inform us of what is happening at a distance. These sense organs are small in size, but important in function. They are so important that we organize great scientific efforts to study their nature and their behavior, and we come together in international conferences to tell of our discoveries, and to discuss our many unsolved problems.

As members of this Conference we are, of course, more concerned with the ear than with the eye, but since these two organs constitute our principal means of perceiving the energy that is transmitted to us from distant objects, the eye and the ear have much in common; and since, as scientists, we are deeply interested in the general laws of nature, we sometimes find it highly instructive to study the sense of

*This research was carried out partly under Contract Nonr-1866(15) between Harvard University and the Office of Naval Research (Project Nr142-201, Report PNR-198), and partly under National Science Foundation Grant G-2668. Reproduction for any purpose of the U. S. Government is permitted.

hearing side by side with the sense of seeing. What we learn from the study of vision can sometimes clarify our study of audition, and vice versa.

The particular problems I shall discuss relate to the manner in which the eye and the ear respond to the intensity of light and sound.

ELECTRICAL EFFECTS.

Both the eye and the ear are what we call transducers; they convert energy from one form into another. Although their principal function is to convert light and sound into nervous energy, they are also able to perform other interesting transformations. Both these sense organs convert the energy of their respective stimuli into electrical energy, as demonstrated by the "electro-retinogram" in the eye and the "aural microphonic" in the ear. These electrical phenomena turn out to be far more complex than was first supposed, but since we now know how to amplify and record the minute electrical potentials generated in the sense organs, we can use these potentials to study the action of the sensory transducers. In the expert hands of my colleague, Dr. Békésy, these methods have revealed important facts about the "electro-anatomy" of the complicated structures in the inner ear.

Not only do the sense organs turn light and sound into electricity, but when we pass electric currents through these organs a sensation results. Volta discovered this fact back in the year 1800, when he first connected some of his newly invented electric cells to metal rods inserted in his ears. Some time later, E. H. Weber did a similar experiment, using his brother as the subject, but when he passed a current through the head, his brother saw a light. This may have been the first instance of the production of what we now call a phosphene. Actually, these early experiments were not very successful. Fairly high D.C. voltages were used, and the result was too painful to bear repeating. With smaller A.C. voltages it is quite possible to make a person hear a tone, or even a radio program. I once filled my ear with salt water and connected the output of a radio set to an electrode immersed

in the liquid. I was able to hear the program, but it was quite distorted until a D.C. voltage was simultaneously applied to the electrode; then the speech and music became very clear. My first thought was that this method of exciting the ear, which I have called the electrophonic effect, might prove an aid to the hard of hearing, but we soon discovered that an electrical stimulus becomes painful before it becomes loud enough to be useful. When this sort of experiment is carried out on a normal ear, with an intact eardrum, the electric current probably does not excite the inner ear directly, but merely sets up audible vibrations in the skin.[5]

In order to excite the cochlea directly, it is necessary to use a patient whose eardrum has been removed. We once investigated 20 ears that lacked tympanic membranes.[6] When stimulated by a sinusoidal electric current, 11 of these ears heard a pure tone, indicating that we may have been stimulating the cochlea directly. Seven of the ears heard only a noise. We may interpret the hearing of a noise as the sinusoidal current stimulating the auditory nerve directly. In these experiments we sometimes stimulated other nerves—the facial nerve and the vestibular nerve—even though we were trying not to do so.

SENSITIVITY.

One of the most amazing things about our sense organs is their sensitivity. The eye is such a sensitive device that the minimal energy that must reach the retina in order for a man to see is a mere half dozen quanta. If the eye were much more sensitive it would probably do us little good, because light energy does not come in smaller than quantal packages.

The sensitivity of the ear is no less surprising. The ear is the most elaborate mechanical system in the human body. At those frequencies where this complex mechanism is most sensitive, a sensation of hearing is produced when the eardrum moves through an amplitude that is less than the diameter of a hydrogen atom. Under these circumstances, the motion of the basilar membrane is even less than that of the eardrum—approximately 100 times less. At the threshold of

hearing, the basilar membrane moves through a minute amplitude which is even less than the wave length of X-rays.

Calculations reveal that the minute displacements needed to excite a sensation of hearing in a good ear are of the same order of magnitude as those that are produced by the thermal agitation of the molecules of the air itself. To the ear with superior hearing, therefore, we may assume that any further increase in sensitivity would be of little use, unless one cared to listen to the banging of the molecules. Like the eye, the ear can perceive almost as small a stimulus as nature can produce.

DYNAMIC RANGE.

Although, in evolving these transducers with their superb sensitivity, nature has pushed the thresholds down to where they are limited by the characteristics of the stimulating medium; nature has not forgotten that useful sense organs must also respond well to high intensities. The dynamic range of the intensities to which these organs react is greater than a trillion to one.

In order to measure such extreme ranges of energy, we commonly resort to the use of logarithmic scales, of which the decibel scale is an example. We are all familiar with the use of decibels for the measurement of sound intensity, but it is interesting to note that the decibel is equally useful as a measure of light intensity. By choosing appropriate reference levels, we have set up a common decibel scale for both light and sound in such a way that interesting similarities between the two senses are at once apparent.[8]

The common decibel scale is shown in Fig. 1. The reference or zero db. levels are 10^{-10} Lambert for vision and 10^{-10} microwatt per square centimeter for audition. Both of these reference levels are near the absolute thresholds of the two senses.

First, we note that the luminance of the sun, our major source of illumination, is about the same number of decibels above threshold as is the intensity of a jet airplane with afterburner, which is probably the loudest sustained man-made noise yet produced. Listening to the noise of such an airplane

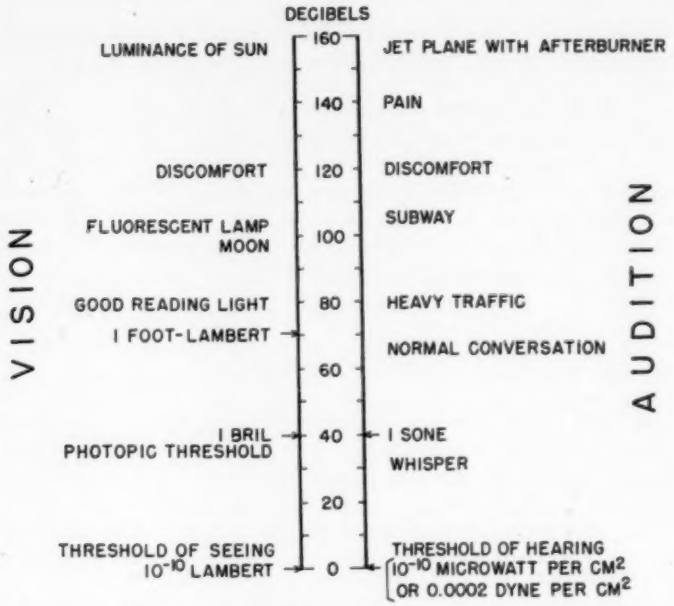


Fig. 1. A decibel scale for light and sound, showing the approximate levels of luminance and of sound intensity produced by various sources, together with a few important threshold levels. The points indicated by arrows are exact levels fixed by definition. The other levels are approximations only.

is hard on the ears, just as looking directly at the sun is hard on the eyes. Both are far above the level where discomfort begins, which is at about 110 to 120 db. on both scales. The comfortable brightness for seeing and the comfortable loudness for listening are both near the middle of these decibel scales, and when stimuli are less than about 40 db. we strain to perceive them.

These decibel scales demonstrate that the dynamic ranges of vision and audition are enormous. The sense organ processes that turn physical energy into nervous energy are such that the output of the sense organ grows with the input over a range of the order of 150 db. In this respect, our sense organs are probably superior to any man-made transducers yet devised.

THE MEASUREMENT OF SENSATION.

Although we use decibel scales to measure the intensity of the stimulus, we must realize that the decibel is not a measure of the sensation produced. By this I mean that a stimulus at 50 db. does not appear to us to be half as great as a stimulus at 100 db. It seems much less than half as great. The law that governs the apparent growth of sensation is not the logarithmic law that Fechner proposed more than a hundred years ago. If Fechner's law were correct, both brightness and loudness would be proportional to decibels. But when a person is asked to compare 50 db. with 100 db. he nearly always says that the 50 db. stimulus looks, or sounds, much less than half as strong. Most people report that the 50 db. stimulus appears to be only about one-fiftieth as great as the 100 db. stimulus.

During the years we have performed many different experiments to try to determine more precisely the law relating the magnitudes of the sensations of light and sound to their respective stimuli. Although different procedures give slightly different results, certain general principles are becoming clear. It is clear that we must replace Fechner's logarithmic law by a quite different formula, for the basic psychophysical law turns out to be a *power function*. For example, when people are asked to make direct quantitative estimates of the subjective brightness of a white light, the magnitude of their estimates is roughly proportional to the cube root of the physical intensity; and to me a most remarkable fact is that approximately the same law appears to hold when people are asked to judge the subjective loudness of a "white noise."

In order to fix a unit for the subjective scales which we use to measure brightness and loudness, we have chosen the stimulus level of 40 db. (see Fig. 1). Thus we define a *bril*, the unit of subjective brightness, as the brightness one experiences when looking at a luminance of 40 db., with the eye dark-adapted. Similarly, the unit of subjective loudness, the *sone*, is the loudness of a 1000-cycle tone at 40 db. (40 phons). On this scale, a loudness of 2 sones sounds twice as great as a

loudness of 1 sone, a loudness of 10 sones is ten times as loud, and so forth.

For a 1000-cycle tone, the standard reference signal whose sound pressure level we use as the yardstick to measure loudness level in phons, the rule is roughly the same. From the pooled results obtained in several different laboratories we have determined that the loudness in sones S is related to loudness level in phons by the formula

$$\log S = 0.03P - 1.2.$$

This formula tells us that in order to change loudness by a factor of two we need to change the stimulus by 10 phons.[9]

The approximate similarity between loudness and brightness is shown in Fig. 2. In both experiments we merely asked observers to assign numbers to the various stimuli (presented in irregular order), and to try to make the numbers proportional to the apparent loudness or the apparent brightness. The brightness experiment was conducted by J. C. Stevens. The fact that the data fall on straight lines in a log-log plot shows that both loudness and brightness are proportional to the stimulus intensity raised to a power.

What we learn from all this is that the loudness heard by a normal ear does not bear a simple one-to-one relation to sound energy, nor to sound pressure, nor to decibels. What we perceive is a sensation resulting from the complex action of the ear and the nervous system, acting as transducers in a manner we do not yet fully understand.

RECRUITMENT.

Let us now discuss another similarity between vision and audition. It concerns the problem of recruitment, a subject of great interest to audiologists. In its extreme form, recruitment is a striking phenomenon, because an ear that shows recruitment may be hard-of-hearing for faint tones and yet hear loud tones with the same loudness heard by a normal ear. In other words, as the intensity is increased, loudness grows more rapidly in some ears than in others.

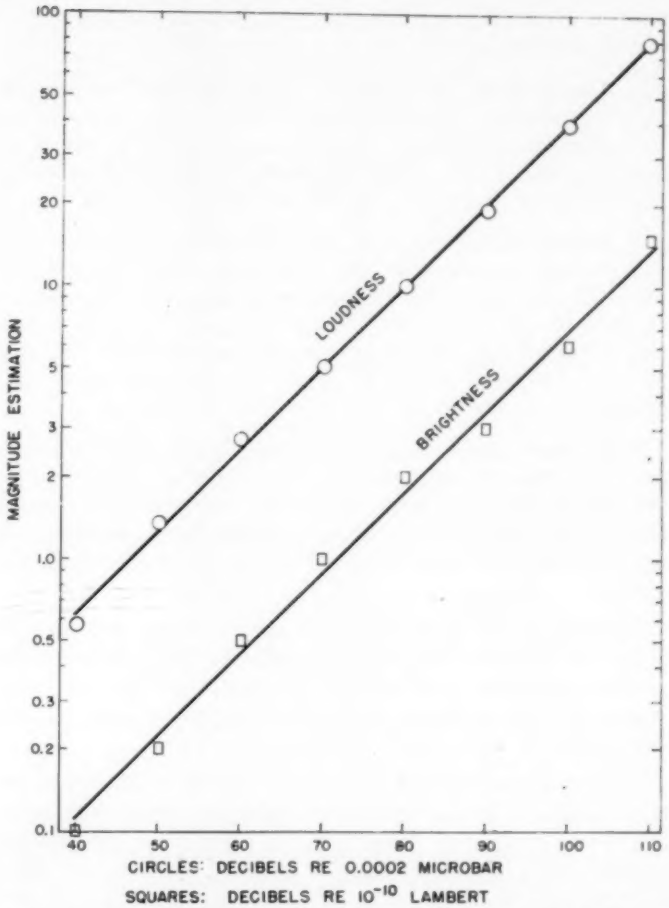


Fig. 2. Median magnitude estimations for loudness and brightness. For loudness each of 32 observers made two estimates of each level of a 1000-cycle tone. For brightness each of 28 dark-adapted observers made two estimates of each level of a white light. The target subtended a little less than 5 degrees and was illuminated for about three sec. at each presentation. The two sets of data have been separated vertically by an arbitrary amount. Hence the ordinate scale is relative only.

Actually we can distinguish at least four ways in which the rate of growth of loudness with intensity can be altered. All four of these are sometimes referred to as recruitment. They are as follows:

1. Recruitment as a function of frequency. The loudness of low-frequency tones grows more rapidly than the loudness of high-frequency tones. Thus, when a tone of 50 cycles per second is only 30 db. above threshold, it may sound as loud as a 1000-cycle tone that is about 50 db. above threshold.

2. Recruitment under masking. The presence of a masking noise raises the threshold for a tone, but when the intensity of the tone is raised above the masked threshold, the loudness of the tone grows rapidly. When the intensity of the tone is raised sufficiently far above the masked threshold, the tone sounds as loud as it would sound if no masking noise were present.

3. Recruitment under fatigue or adaptation. Exposure to a loud noise may temporarily raise the threshold of the ear by as much as 60 db. or more. In an ear suffering from such a temporary hearing loss, loudness grows more rapidly with intensity than in an ear that has not been fatigued by a loud sound.

4. Recruitment in pathological ears. There are diseases of the ear that can cause hearing losses for faint tones without greatly impairing the loudness perception of intense tones. We know that this kind of recruitment exists, but we still have much to learn about its causes.

What concerns us here, however, is not so much the causes of recruitment as the fact that analogous phenomena occur in vision. This is an important fact which we should keep in mind when we are tempted to theorize about the mechanisms underlying recruitment. The phenomenon is by no means limited to the auditory sense.

Each of the four classes of recruitment which we have already discussed has its parallel in vision. Let us consider each of them in turn:

1. As is true for loudness, brightness varies with intensity

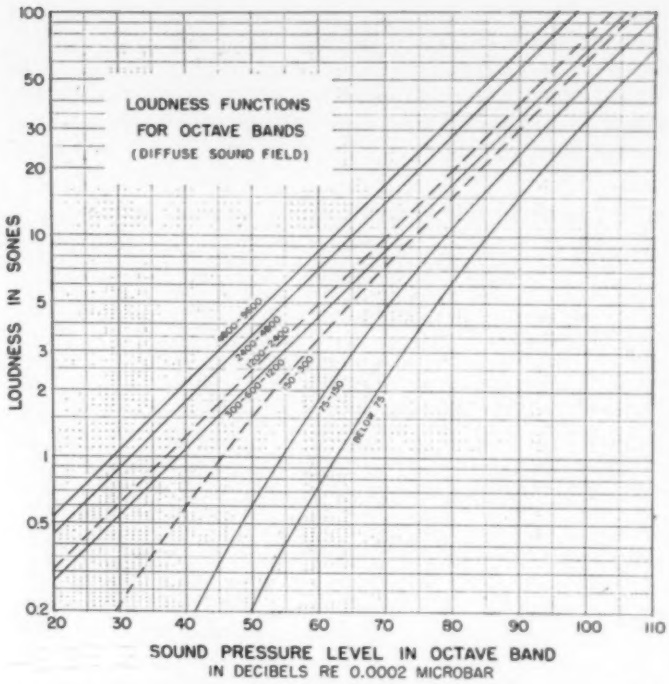


Fig. 3. The relation between loudness and sound pressure level for octave bands.

more rapidly for some wave lengths than for others. Thus a red light appears to grow brighter at a faster rate than does a green light when the intensity is raised above threshold.

These basic facts are shown in Figs. 3 and 4. Fig. 3 shows how the loudness of various octave bands of noise grow with sound pressure level. This chart was prepared to facilitate the calculation of the total loudness of a noise on the basis of octave-band measurements.[10] The total loudness turns out to be the loudness in sones of the loudest octave band plus 30 per cent of the sum of the loudnesses in all the remaining bands.

The rapid growth in the loudness of the low-frequency

bands in Fig. 3 has a rough parallel in the rapid growth of the luminance of red light (650 mμ) shown in Fig. 4. *Luminance,* when measured in decibels, is analogous to our concept of *loudness level,* and is proportional to the logarithm of the subjective brightness. The data in Fig. 4 are from estimates made by Weaver.[11]

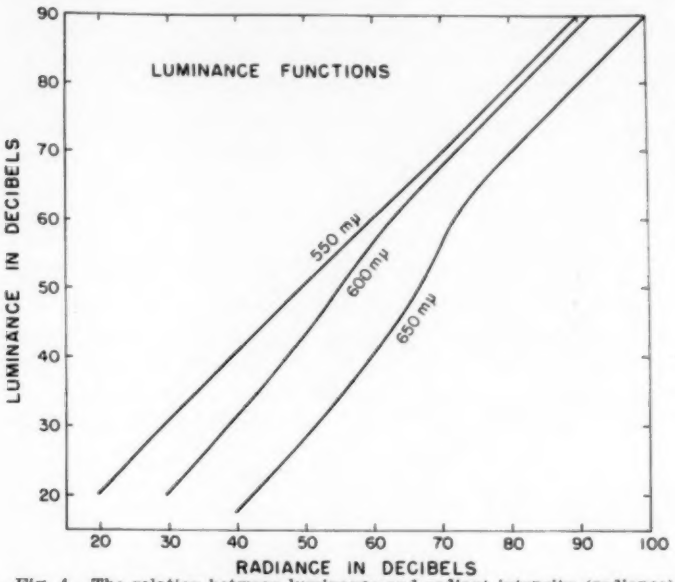


Fig. 4. The relation between luminance and radiant intensity (radiance) for three different wave lengths of light ranging roughly from yellow to red.

2. Masking in hearing has its analogue in what is called *glare* in vision. In the presence of a source of glare, the absolute threshold of the eye is raised, but when the stimulus is made more intense than the glare the apparent brightness of the stimulus grows to the value it would have even though the glare were not present. Thus *masking* recruitment occurs in both vision and audition.

Recruitment curves under masking and glare are shown in Figs. 5 and 6. As shown in Fig. 5, masking raises the

threshold of a tone, but as the level of the tone is raised its loudness soon reaches the value it would have if the masking noise were not present.[7] In Fig. 6 we see that a very similar effect takes place in the eye. Bright glare patches were placed beside a stimulus target viewed with one eye, and the brightness of the target was matched by a target in the other eye,

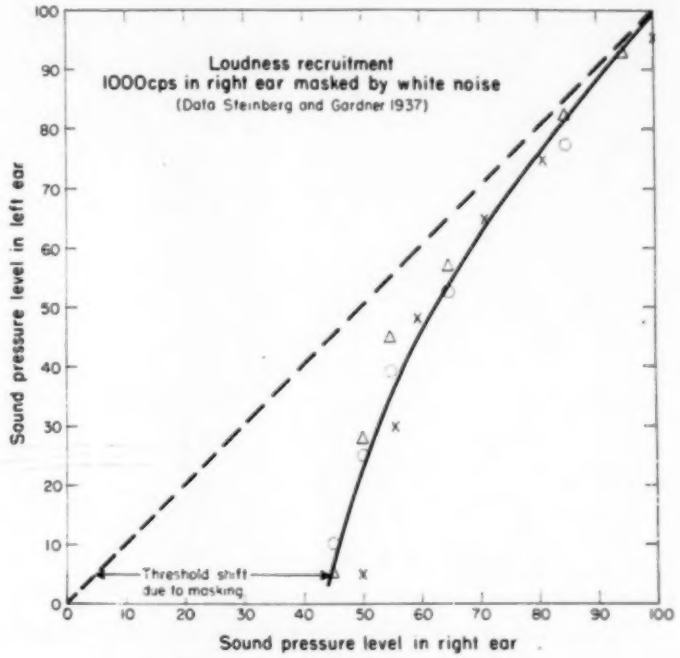


Fig. 5. Loudness recruitment under masking.

which was not subjected to glare. The unfilled points are from an experiment by Diamond.[3] The filled points show our attempt to complete the curves by determining the threshold shift under the same stimulus conditions.

3. We have also found that in the eye that has been exposed to a strong light brightness grows more rapidly with intensity

than it does in the eye that is dark-adapted. Exposure to strong light raises the absolute threshold of the eye in much the same way that exposure to intense sound raises the threshold of the ear. There are many important differences here, but in both cases the perception of intense stimuli remains more nearly normal than the change in threshold would lead

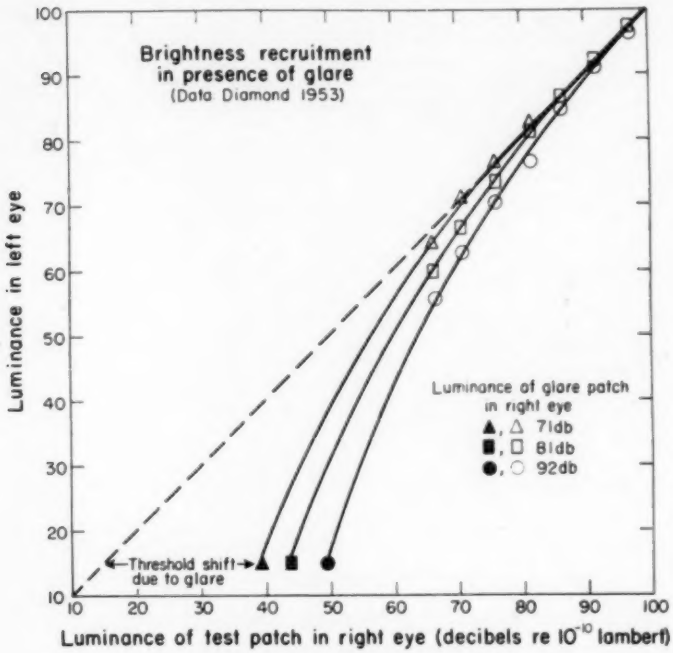


Fig. 6. Brightness recruitment under glare.

us to expect. In other words, brightness recruitment occurs in the light-adapted eye, just as loudness recruitment occurs in the ear that has been temporarily fatigued.

These relations are shown in Figs. 7 and 8. Actually the effects of exposure to intense sound are quite variable, and so are the resulting recruitment curves. The particular curve

shown in Fig. 7 was obtained by exposing one ear to a wide band of noise for 32 minutes at a sound pressure level of 130 db.[2] Loudness balances were then made between the exposed and the normal ear at five frequencies between 1000 and 1700 c.p.s. The points in Fig. 7 represent the averages for the five frequencies. We see that, although the threshold was

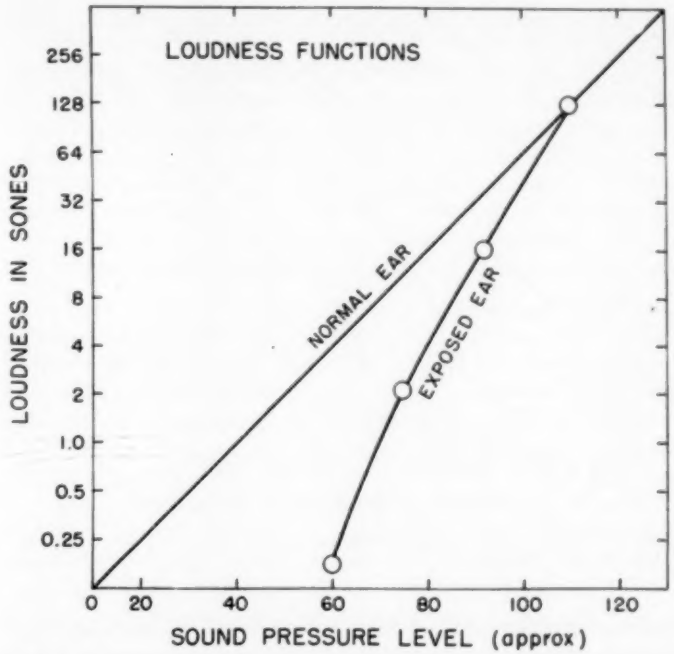


Fig. 7. An example of a temporary hearing loss produced by exposure to an intense noise. The exposed ear shows loudness recruitment.

raised by the noise exposure, the loudness heard by the exposed ear was essentially normal for tones at an SPL of about 110 db.

The effects of exposing the eye to bright light are shown in Fig. 8. These data show that over the low luminance levels the light-adapted eye shows recruitment, *i.e.*, the curves are

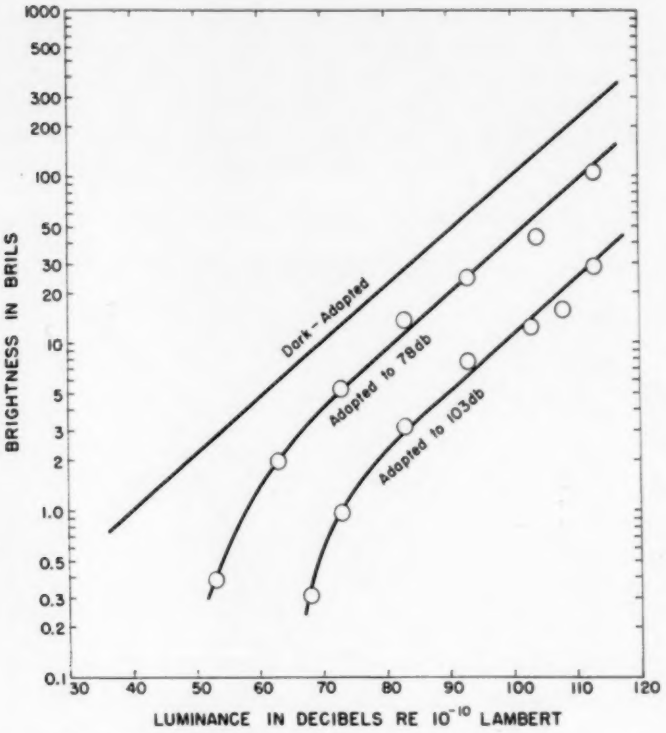


Fig. 8. Showing how light adaptation leads to brightness recruitment at low levels of luminance.

steep at the low end. On the other hand, there is an important difference to be noted. In the eye the recruitment is not complete. In other words, after our eyes are adapted to a bright light, all lights look dimmer than they would if we were dark adapted, but the effect on lights near threshold is relatively greater than the effect on the higher levels.

It should be mentioned that the positions of the curves through the points in Fig. 8 were determined in an experiment[1] involving brightness balances between the two eyes, with one eye dark adapted and the other light adapted. The

points themselves were obtained by J. C. Stevens, who asked groups of 18 light-adapted observers to estimate the apparent brightness of various stimuli.

4. Certain pathological conditions in the eye can also raise the absolute threshold without causing a correspondingly large change in the apparent brightness of intense lights. I have recently had the opportunity of examining a case of what is called congenital stationary night-blindness. The patient was a young man who apparently saw quite normally under high illumination (except for a slight myopia) but who saw nothing when the luminance was below about 40 db. He has a brother and a sister who have similar deficiencies. So far as I know, brightness perception in these cases has not been explored as thoroughly as loudness perception in ears that show auditory recruitment, but the fact that visual recruitment seems to exist in certain abnormal eyes is interesting evidence of a further functional similarity between the eye and the ear.

In these remarks I have stressed some of the functional similarities between vision and audition. It would be equally possible, of course, to stress the differences between these two remarkable sense organs, for there are indeed important differences. Even where their behavior is similar, the underlying mechanisms may of course be quite different. Not all the problems faced by audiology are similar to the problems that arise in the visual sciences, but there are enough parallels between the general laws of vision and those of hearing to make it highly profitable for us to study the eye in order to learn more about the ear and vice versa.

Let us consider one final matter, concerning another type of loudness growth—a phenomenon that could be regarded as a form of recruitment, but one that seems to have no parallel in vision: dependence of loudness on the band width of a sound. From recent studies of this phenomenon there emerges the important concept of a critical band width.

Figs. 9 and 10 show examples of data from a study by Zwicker, Flottorp and Stevens.[12] In these experiments the listener made a loudness balance between a comparison sound and either a complex of four pure tones (all at a constant

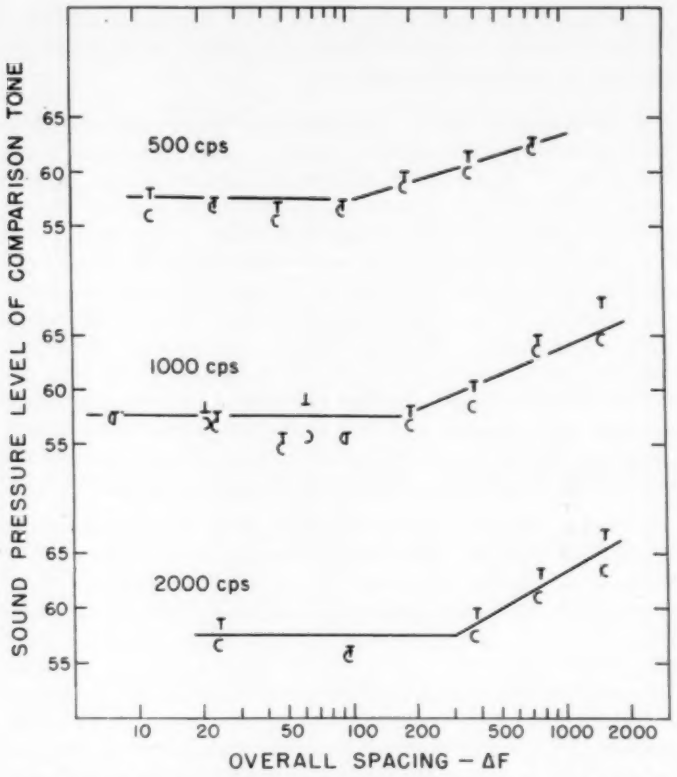


Fig. 9. Showing how loudness depends on the overall spacing ΔF of a four-tone complex. The spacing of the tones was uniform about the center frequency, indicated on each curve. Each point is based on loudness balances by 16 to 22 listeners. The symbol "T" means that the single comparison tone was adjusted; "C" means that the complex was adjusted. The lines through the data have a break at the frequencies determined by other critical-band determinations.

level) or a band of noise. In Fig. 9 we see that, as the spacing between the tones is increased, the loudness of the complex remains essentially constant until the over all spacing $\triangle F$ reaches a critical value. Beyond that point the loudness grows as the spacing is increased. In other words, there is a kind of recruitment. The critical value of $\triangle F$ depends on the frequency region in which the tones lie.

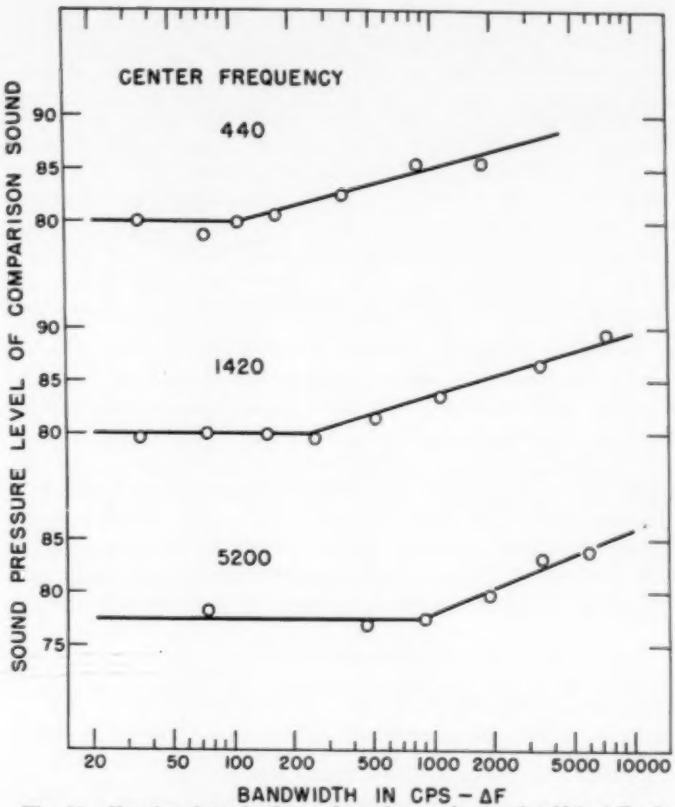


Fig. 10. Showing how loudness depends on the band width ΔF of a noise of constant sound pressure level. The lines through the data have a break at the frequencies determined by other critical-band determinations.

In Fig. 10 we observe a similar effect when we increase the width of a band of noise whose over all sound pressure level is held constant. Beyond a critical point the loudness increases with band width, but as long as the energy lies within a critical band all that seems to matter for the determination of loudness is the sound pressure level of the noise. In fitting the lines to the data in Figs. 9 and 10 we placed the break between the horizontal and the rising segment at the points

dictated by measurements made in three other types of experiments. In other words, four different techniques have converged on the determination of this critical band width.

The critical band width thus measured turns out to be about 2.5 times as large as the critical band width originally proposed by Fletcher,[4] which was based on a different kind of experiment; nevertheless, in their dependence on frequency both Fletcher's band and this new band follow much the same course. The width of the new critical band is about 90 c.p.s. at the low frequency end of the spectrum. Near 1000 c.p.s. the widths is about 170 c.p.s., and near 10,000 c.p.s. it is about 2400 c.p.s.

Not only does this critical band width emerge in different kinds of experiments, but it also seems to be closely related to other important auditory functions. For example, each critical band occupies about the same width on the basilar membrane. Each band contains about the same number of just noticeable differences in pitch, and each contains roughly the same number of mels (units of subjective pitch). We have reason to believe, therefore, that this critical band width may come to hold an important place in auditory theory.

REFERENCES.

1. Craik, K. J. W.: The Effect of Adaptation on Subjective Brightness. *Proc. Roy. Soc.*, B128, 232-247, 1940.

2. Davis, H.; Morgan, C. T.; Hawkins, J. E.; Galambos, R., and Smith, F. W.: Temporary Deafness Following Exposure to Loud Tones and Noise. *Acta Oto-Laryngol.*, Stockh., Suppl. 88:1-57, 1950.

3. Diamond, A. L.: Foveal Simultaneus Brightness Contrast as a Function of Inducing- and Test Field Luminances. *Jour. Exp. Psychol.*, 45:304-314, 1953.

4. Fletcher, H.: Auditory Patterns. *Rev. Mod. Phys.*, 12:47-65, 1940.

5. Flottorp, G.: Effect of Different Types of Electrodes in Electrophonic Hearing. *Jour. Acoust. Soc. Amer.*, 25:236-245, 1953.

6. Jones, R. C.; Stevens, S. S., and Lurie, M. H.: Three Mechanisms of Hearing by Electrical Stimulation. *Jour. Acoust. Soc. Amer.*, 12:281-290, 1940.

7. Steinberg, J. C., and Gardner, M. B.: The Dependence of Hearing Impairment on Sound Intensity. *Jour. Acoust. Soc. Amer.*, 9:11-23, 1937.

8. Stevens, S. S.: Decibels of Light and Sound. *Physics Today*, 8(10):12-17, 1955.

9. Stevens, S. S.: The Measurement of Loudness. *Jour. Acoust. Soc. Amer.*, 27:815-829, 1955.

10. Stevens, S. S.: Calculation of the Loudness of Complex Noise. *Jour. Acoust. Soc. Amer.*, 28:807-832, 1956.

11. Weaver, K. S.: A Provisional Standard Observer for Low Level Photometry. *Jour. Opt. Soc. Amer.*, 39:278-291, 1949.

12. Zwicker, E.; Flottorp, G., and Stevens, S. S.: The Critical Band Width in Loudness Summation. *Jour. Acoust. Soc. Amer.*, 29:548-557, 1957.

AN ATTEMPTED SYNTHESIS OF PSYCHO-ACOUSTIC TEST DATA IN THE AUDIOLOGICAL CLINIC.

J. Donald Harris,

U.S.N. Medical Research Laboratory,

New London, Conn.

More than a dozen separate tests, based upon as many different principles, have been proposed in recent years to assist in distinguishing "conductive" and "perceptive" deafness. Many of these tests, even the classic Weber and Rinné examinations, do succeed well in making this first distinction; and some, such as careful BC audiometry, can often give a quite precise idea of the relative components in mixed deafness.

One notes that the terms "conductive" and "perceptive," in past decades thought useful and clearcut, are today eroding rapidly. A variety of causes and sites of "conductive" lesion render that term a catch-all with less precision than now required. Mygind and others even speak of "conductive" lesions within the cochlea; here the original division has broken down completely. On the other side, it is now clear that the category of "perceptive" deafness has even less explicit meaning. It is relatively useless to lump all disorders of the auditory mechanism from perilymph to cerebrum under one compendium phrase, if one can be more specific.

It is fair to say that one of the advances which has assisted otologists to be more specific has been the introduction into the clinic of a great deal of psycho-acoustic thought by the otologists and by supporting persons. There is implicit here a whole new concept in otological diagnosis, namely, that since a particular physical disorder of pathology in certain

patients can be shown to cause atypical behavior on a certain psycho-acoustic test, that test may be useful in throwing light on the diagnosis in another patient whose purely physical examination yields ambiguous concepts as to the locus or etiology of the dysfunction.

Otologists have been impelled to seek for such inferential light in diagnosis of "perceptive" disorders because for the most part, these tissues in the auditory mechanism are, and must remain, inaccessible to the otoscope, and even to the exploratory surgery of the tympanum, in which such giant strides are currently being taken; however this may be in the future, it now seems possible with psycho-acoustic assistance to go beyond the rather coarse conductive-perceptive dichotomy and begin to break down the latter category into at least three subdivisions, neural, end-organ, and cochlear (non-end-organ). The neural category can of course be broken down further in some patients, and there is even the hope that in the future an attack can be made on the very indefinite category "cochlear (non-end-organ)."

Having thus placed psycho-acoustic thinking in perspective as a part of the armamentarium of the otologist, it remains to consider what test or tests could, or even should, be available in the complete clinic. I should like to draw your attention to: *1.* a brief discussion of how some of the present psycho-acoustic tests have come to be suggested; *2.* turn to one rational line of reasoning as to how a complete and mutually exclusive battery of such tests might be constructed; *3.* how a "profile" of test results may correlate higher with diagnosis than results from any one or two tests.

It will be instructive first to list a few of the psycho-acoustic tests or criteria which have been proposed for the clinic:

AC-BC Audiogram, Level and Shape.
AC-BC Differential.
Threshold Uncertainty Interval.
Shape of Speech Intelligibility Function.
Loudness Balancing, and Shape of Recruitment Curve.
Loudness-Modulation Sensitivity.

Loudness-Increments Sensitivity.
Sensitivity for Loudness by Constants.
Pitch-Modulation Sensitivity.
Pitch-Memory Sensitivity.
Perstimulatory Adaptation.
Post-Stimulatory Adaptation and Fatigue.
Temporal Integration.
Pure Tone Distortion Threshold.
Masking of Speech and Pure Tones.
Intelligibility of Speeded and Reverberant Speech.

That this list is by no means complete is indicated by the fact that each of these tests has a bibliography of from several to dozens of items. In one recent review of recruitment, for example, over a dozen tests of recruitment and/or closely allied topics were listed. For another example, the field of loudness discrimination is burgeoning, and new tests not listed above are appearing all the time.

A number of these tests have been worked up for entirely non-clinical purposes, and their present audiological relevance has arisen mainly by chance—witness the work of Békésy, Hood and others. Some writers have created audiological tests on the basis of some good guess as to the nature of the disorders and the capacities of the defective ear—Lüscher, Langenbeck, Huizing, Hood again, Miskolczy-Fodor, and others. What happens is that someone suddenly has an idea that pitch discrimination, reverberant speech, or auditory masking, may assist in otological diagnosis, and a number of patients (it is the rule to use far too few for any solid reliability) are given the new test, with the result that the original author is quite enthusiastic about its nosological meaning.

Following this, another laboratory or clinic, changing experimental conditions freely, gives what is said to be the same test and finds conflicting or ambiguous results. This exact sequence has happened with tests of residual auditory fatigue, with masking, with intensity discrimination, with temporal integration, and with perstimulatory adaptation, to name several recent examples. At the present time, all workers are convinced that the principle is sound, that such

tests will ultimately prove valuable in diagnosis, but no general agreement is had on any specific test.

The difficulty with these multi-various test possibilities is that they are not mutually exclusive, or in other terms, they are not factorially pure auditory traits. A pure auditory trait is assumed to have a direct relation to the way the ear transduces and transforms the acoustic stimulus into cochleoneural events. A particular psycho-acoustic test, on the contrary, may be far from pure, partaking of so many primary auditory abilities that its results become ambiguous. This means that a patient's atypically poor performance on this test cannot throw any light on the condition of specific tissues or processes in the auditory mechanism.

As a result of studies at this laboratory* we now know, for example, that pitch discrimination, long thought to be a primary auditory ability, is actually broken down into two factors, pitch-memory and pitch-modulation. A normal ear may be very good on one of these traits but only average on the other. A defective ear on the same head may show a different pattern. For another example, the detection of speech in noise is found *not* to be related to the detection of a sine wave, or bands of noise, or clicks, or Gaussian pulses, but is factorially complex with a large specific component of its own with alliance to factors of pitch and temporal patterns. For a third example, we suspect speeded-up speech to be factorially fairly pure, by analogy, in the field of intelligence testing, to the primary mental ability of verbal facility.

Only when a test is factorially pure can there be a chance of its representing fairly elementary operations of the auditory mechanism, so that if an ear performs badly on that test, the source of trouble in the auditory mechanism can be pinpointed. A factorially complex test will almost hopelessly becloud the clinical issues. We have already seen this happen for loudness discrimination, as I shall amplify later.

I feel it is now possible to take a completely rational ap-

*MRL Report No. 57-4.

proach to the problem of audiological tests, using the technique of factor analysis as mentioned. Our work using this approach in pitch discrimination, loudness discrimination, contact detection, and speech reception has been summarized.* We can now suggest "tag tests" for two pitch factors, three loudness factors, and two or more distorted speech factors.

As a paradigm, let us examine the matter of psycho-acoustic tests for susceptibility to acoustic trauma. Many industrial physicians and audiologists have proposed a brief exposure to moderate sounds, and a single threshold-shift index assigned to each man; but these proposed tests have used exposures ranging from 0.4 sec. to 10 min. frequencies over a broad range including white noise and clicks, and a wide variety of other dissimilarities exists. Should one give all these tests? The technique of factor analysis provides a means to reduce them to the fewest number which will sample the whole field of temporary threshold shift. Large groups of subjects would serve for the following program:

a. Outlining the area with a dozen or more specific test ideas. One would include among other things tests of aural overload, latent damage, short-duration adaptations, etc.

b. Tape-recording these preliminary tests with appropriate psycho-acoustic equipment and psycho-physical techniques acceptable by today's standards.

c. Trying them out for reliability, and standardizing them on pilot groups.

d. Tape-recording a final battery of tests and presenting to at least 200 subjects with normal ears.

e. Inter-correlating and factor analyzing.

f. Interpreting the data in terms of primary auditory abilities.

The smallest number of tests which would explain variance

*MRL Report No. 57-4.

among subjects would then be cast into a predictive battery and administered to men destined for high noise areas.

Such an approach, with tapes widely distributed in several countries, would bring order into a confused field and bring final predictive success much closer to reality.

Exactly the same sort of statistics-plus-psycho-acoustics is suggested for the otological clinic, except that the final winnowing of standardized pure tests in any area, such as loudness discrimination, would be further used to explore the diagnostic significance of each of the several primary abilities within the broad area of loudness discrimination.

It may be well to explain this last point here. Lüscher and Zwislocki first succeeded in using a measure of loudness discrimination as a clinical index of recruitment, and subsequent data by Neuberger and others from that laboratory have convinced us that, in general, the otological conditions associated with recruitment are also associated with improved sensitivity for amplitude-modulation. On the other hand one recalls Denes and Naunton's conclusion, exactly opposite from Lüscher's, that near threshold the recruiting ear shows a relative improvement in loudness sensitivity. Note, however, that Denes and Naunton used a "constants" method, with an interval of silence between two tones to be judged; it should now be clear why we feel that the techniques of loudness sensitivity by the "constants" method and the "amplitude-modulation" method *both* are applicable to the defective ear, and can both be used to assist in diagnosis. Rather heavy criticisms of each method have been made by the other school, on the basis that "amplitude-modulation" produced side-bands to blur the "quality" of the tone or that the "constants" method yields an incongruously poor sensitivity at higher intensities, but it seems that such criticisms are beside the point; furthermore, it is not at all necessary to survey the several inconclusive reports and the several apparently contradictory reports, throw one's hands up and simply await the appearance of further inconclusive and inconsistent reports.

The difference in results between the "constants" and the

"modulation" tests goes far beyond the elementary notion that the exact value of a DL depends upon the techniques used to measure it—here we are not dealing with the same auditory trait. In "amplitude-modulation" the ability is rendered more acute by whatever otological conditions cause recruitment, and this improvement exists at about equal strength over the whole range of sensation level. On the other hand, in "constants" the ability is rendered more acute but *only in the region of rapid recruitment.* A particular type of pathology may affect one trait in one way and the other in a quite different way.

With this thought in mind we are better able to assess the several reports which have appeared since the original validating papers. These reports are mutually informative; not contradictory.

I have said perhaps enough to point out how we expect to generate newer and more meaningful ("purer") tests for auditory traits with continued effort. Particularly, we feel that the areas of speech, of temporal patterning and rhythms, of masking and contact detection, of auditory fatigue and adaptation, and the area of inter-factor relationships must be explored.

I now direct your attention to the third point, that in a particularly difficult diagnostic situation one may look at not only the results of tests one by one, but also at the interrelations among the sets of results. It is the pattern, or profile, that may well be the really important clue. For example, presbycusis is often characterized by lowered BC, a certain slope of audiogram, absence of recruitment, distortion of temporal integration, and lack of resistance to speeded speech. If one looked at only one or two of these tests an uncertainty might exist, but the total picture becomes clearer when the test profile is considered. For another example, we have published data showing that improved loudness discrimination need *not* be paid for with a deteriorated pitch discrimination; but that this is *always* true with acoustic trauma.

We have a patient with advanced otosclerosis who exhibits a breakdown in "intensity-modulation" at weak levels, but whose loudness by "constants" remains quite good. We have a patient with a pure conductive loss by BC, perstimulatory adaptation, and a half-dozen other signs, but who exhibits straight-line recruitment, disturbance of temporal integration, and diminished intensity-modulation. We have a patient with a sudden unilateral loss above 500 c.p.s. caused probably by a vascular spasm, with no AC-BC differential, extreme disturbance of temporal integration but with delayed and slight straight-line recruitment. We have several patients with quick and complete recruitment, disturbance of temporal integration, no AC-BC differential, and history of acoustic trauma, but some do and some do not show unusual perstimulatory adaptation. We have a patient with mixed deafness which would be considered purely conductive except that extremely distorted temporal integration reveals a minor cochlear lesion. We have a patient with high tone cochlear deafness probably familial, with quick straight-line recruitment which indicates that cochlear lesions in addition to acoustic trauma and Ménierè's disease can distort temporal integration.

It is no trouble to explore the protocols of patients in the literature from the practices of Palva, Bocca, Fournier, and also dozens of otologists in this country, who are accustomed to giving several psycho-acoustic tests where indicated or where possible, to find similar instances.

Such of our data as seem internally self-contradictory we regard as among our potentially most powerful results. They are at least not due to scanty sampling, since we are able to schedule both our civilian and our military patients for six to ten hours or more if needed. We regard as in the pioneer stage only, the concepts of factorially pure auditory abilities and tests for them, and of the correlation between pathology in any tissue and a concomitant set of results on a multifactor test battery. Our successes have been limited and have come slowly, but fortunately they have appeared at a rate just sufficient not to extinguish our conditioned drive.

TRIPLET TESTING AND TRAINING—AN APPROACH TO BAND DISCRIMINATION AND ITS MONAURAL OR BINAURAL SUMMATION.

HENK C. HUIZING and M. TASELAAR,

University of Groningen,

The Netherlands.

Recently the principle of what may be called qualitative speech audiometry has excited the interest of some investigators. Kruisinga[1] studied the phenomena of phoneme confusion in cases of abrupt deafness, and he also investigated, in various types of hearing impairment, the change in intelligibility of the spoken voice as compared to that for the whispered voice.

Guberina[2] examined the residual articulative function of impaired ears in discrete octave bands by using adequately chosen logatoms as test material.

The great difference in change of the articulation curve for spoken voice as compared with that for whisper in different types of hearing impairment focused our attention to the significance of the test material in a qualitative sense. This gave us the idea of measuring band articulation, while retaining intelligible speech as test material. The great redundancy, which characterizes normal speech under favorable conditions makes it possible to use narrow band speech which is still intelligible.

By means of suitable filters we are able to narrow the band width more and more until we approach the threshold of intelligibility for the normal ear. To cover the whole tone scale by subsequent frequency bands, three different samples of speech are necessary, *i.e.* a low, a medium and a high quality of speech. Fig. 1 shows the width of each of the three frequency bands equally weighted as to intelligibility for the Dutch language. The filter characteristics are given in Fig. 2.

This split-up of the tone scale in three equivalent parts, with conserved intelligibility in each presents the possibility

of obtaining insight into a patient's partial discrimination ability. We need only to determine his sentence intelligibility or his spondee articulation score at his most comfortable level.

The application of this triplet audiometry on hard of hearing individuals leads to the following conclusions:

1. Triplet audiometry provides more information about the

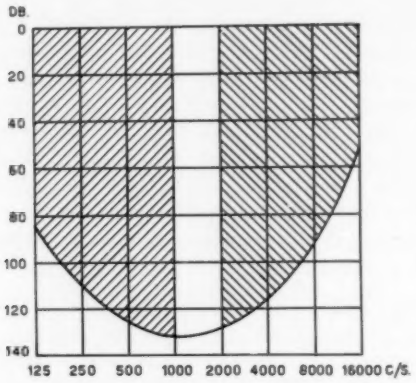


Fig. 1. Triplet frequency bands equally weighted as to intelligibility for the Dutch language.

residual discrimination ability of a patient, especially as to its distribution along the tone scale.

2. Depending upon the type and the duration of his hearing impairment, a patient's discrimination ability may be based upon a normal distribution of discriminative capacity along the whole tone scale, or it may be that his residual discriminative power is mainly concentrated in a rather narrow band of frequencies. The latter may be the case despite the fact that his threshold audiogram still covers the whole tone scale.

3. In cases of long standing deafness with a non-uniform threshold loss in general, the main contribution to intelligi-

bility is given by that part of the tone scale in which the threshold loss is smallest. Depending upon the shape of the threshold audiogram the cerebral patterns of the patient may be low tuned, medium tuned or high tuned. It appears that the shift in energy balance between the various frequency bands is closely related to the category of listeners to which the patient belongs.

4. Triplet speech audiometry provides useful information

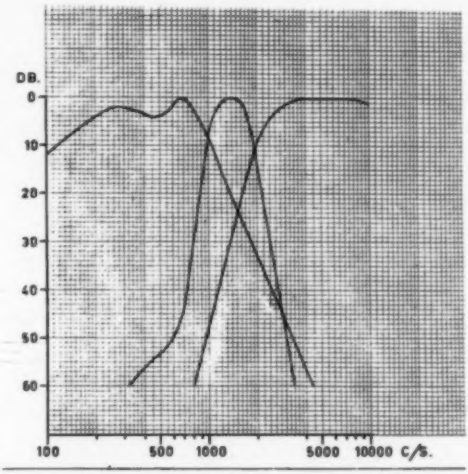


Fig. 2. Filter characteristics used to realize a triple speech sample in equally weighted frequency bands (low, medium and high).

regarding the fitting of a hearing aid on an analytic basis. The analysis may tell us whether it is advisable to try to restore more or less the normal energy relations along the whole tone scale, or that preferably we should confine ourselves to a more or less selective amplification of that part of the tone scale in which the patient's discrimination skill has been kept up.

5. In connection with band discrimination analysis we

direct attention to two-channel amplification. To the credit of some data in the literature,[3] and also on account of our own recent experiments, it may be expected that in certain cases of hearing impairment the intelligibility may be markedly improved when the amplified speech is given through two separated selective bands in binaural stimulation. For this purpose the distribution of discrimination skill along the tone scale must be known for each individual ear.

A double channel system involves a greater flexibility in adjustment of the relative amplification of each frequency band, as well as to the choice of the band for each separate ear. It says that a dual set of hearing aids may give a better opportunity of controlling "power" against "discrimination". It is rather this power discrimination relationship which is frequently disturbed due to non-uniform hearing loss, and often is hard to rectify by a single circuit.

If in the near future we succeed in introducing the binaural aid, the triplet band data of each ear separately will give us useful information for obtaining an optimum improvement.

6. In young children with retarded speech development the triplet discrimination test lends itself principally to the detection of high tone or medium tone deafness (sloping or basin shaped audiograms).

7. Finally this test may tell us whether it is advisable to start an auditory training program in connection with the use of a hearing aid; and if so, what kind of program should be preferable. In this connection it should be emphasized that the triplet system lends itself to selective training in each of the three bands mentioned.

REFERENCES.

1. Kruisinga, R. J. H.: Thesis Groningen University, Nov., 1955.

Huizing, H. C., and Kruisinga, R. J. H.: *Acta Oto-Rhino-Laryngol.*, Belgica, 10:345, 1956.

2. Guberina, P.: *Revue de Laryngologie*, Jan., 1956.

3. Bocca, E.: The Laryngoscope, 65:1164, 1955.

EFFECTS OF AMBIENT NOISE ON SPEAKER INTELLIGIBILITY OF WORDS AND PHRASES.*

JOHN J. DREHER,
and
JOHN J. O'NEILL,
The Ohio State University Research Foundation,
Columbus, O.

I. INTRODUCTION.

Some years ago it was noticed that a speaker with normal hearing would unconsciously raise the level of his voice to compensate for the level of noise in which he was speaking. This phenomenon was incorporated in the development of the "Lombard," or "voice-reflex" test, which was used to detect malingering in the testing of hearing. It would be supposed that as the ambient level of noise for a speaker became greater there would be some changes in his intelligibility. Hanley and Steer[2] found that untrained speakers, in the presence of noise, tended to reduce their rate of speaking, prolong syllables, and speak with greater intensity as the noise increased. Subsequently, Pickett and Kryter[3] concluded that as a speaker increased his vocal intensity from normal level to a shout, his intelligibility decreased at the higher levels. It is noted here, however, that their increases in vocal intensity were accomplished by raising the voice in quiet surroundings, technique that may result in speech with characteristics different from those produced by a speaker actually fighting noise. It was not unreasonable to suppose that speech produced in noisy surroundings might actually incorporate some compensatory factors in it, and that within a certain limited range it might be more effective for a listener under poor reception conditions.

If it could be shown that some of these changes actually

*This study was done under Contract No. AF 19(604)-1577 with Operational Applications Laboratory, Bolling AFB, Washington 25, D. C. (Report AFCRC TN 57-52, ASTIA Document No. AD-110-068.)

act to increase intelligibility, we might anticipate a device that would exploit this fact. For instance, a certain amount of masking might be introduced into the speaker's ears by means of a pre-calibrated device attached to his headset. The listener, who would not be exposed to this noise, would, however, hear the improved output at his end of the link, and the importance of such a device would be obvious in air traffic control and other military voice communication applications.

Inasmuch as automatic volume control is an integral part of modern transmission equipment, the accompanying increase in vocal intensity noted in the "voice reflex" reaction cannot properly be considered as one of the factors legitimate to test. Consequently, the loudness (intensity) changes in the voice were compensated for in the construction of the test, a realistic stricture in view of the eventual application of voice through a limited system.

II. EXPERIMENTAL DETAILS.

A. General Plan—The Lombard reaction, while a general one, varies in magnitude as do other voice measures with individuals; therefore, the plan of the study was to get the reaction of several speakers under several levels of masking at the ear, comparing intelligibility of the speech so produced against a constant background of masking noise with a large number of listeners. To avoid attempts on the speakers' parts to control their vocal level, none were informed of the voice reflex phenomenon.

B. Speakers and Stimulus Materials—Fifteen university students, three male and twelve female, served as speakers. All were of general American dialect and inexperienced with voice recording methods.

Recordings were made with an Altec 21-C condenser microphone positioned along the speaker's cheek with the button behind the corner of the mouth, out of the breath stream. During recording each speaker wore a pair of PDR-8 headphones. These headphones fed the taped output of a Grason-Stadler White Noise Generator (Model 455-B) from an Ekotape recorder to the ears of the speaker. No feedback of the

speaker's voice was provided by the headset, his monitoring being done only by internal sidetone.

Stimulus materials consisted of five sets of spondee words and five air traffic control sentences at each of five recording levels for each of 15 speakers. All words and sentences were different for each speaker at each level; that is, he never had the same material to read more than once during his entire recording stint. The purpose of this was to prevent any accommodation to the phraseology itself. Thus, each speaker read, in all, 25 spondees and 25 sentences. His initial recording was made by wearing a dead headset, and his relatively low resultant voice level (65 db., C scale, one meter distant) served as the base level for the other four recording conditions. He then recorded, in order, stimulus materials with 70, 80, 90, and 100 db. of noise at the entrance to the ear canal. To prevent accommodation to the masking level the noise was on in the headset only during actual production of the speech, the appearance of the noise serving as a cue for the speaker to record. As soon as the test item was read, the noise was turned off for a given length of time to allow for listeners' written response. Three seconds were allowed after each test spondee, eight seconds after each test sentence. A push button operated by the experimenter controlled the noise in the speaker's headset.

The order of the test material was rotated in such a manner that listeners for each of the recorded conditions heard all test words and sentences, since the final test tapes were arranged to present: *1.* all speakers' recordings in quiet; *2.* all speakers' recordings in 70 db. of noise, and so on, five tapes in all.

After this collation of materials according to levels, all tapes were re-recorded through a General Electric BA-5A limiting circuit. It is pointed out here that peak clip limiting was not employed. The operation of this particular instrument is of broadcast quality, set for an attack of 100 microseconds and 0.6 second for 90 per cent recovery. The purpose of such limiting was two-fold: first, to remove the effects of increased vocal intensity from the experimental situation;

second, because such type of intensity reduction is also employed in aircraft broadcast circuits. The total range of limiting involved in these tapes amounted to approximately 10 db.

After re-recording, an overlay of wideband random noise was recorded on the test tapes. The noise overlay was recorded at a level of 4 db. lower than the peak values of the speech signals, both speech and noise being read on the same VU meter. As a check, the completed tapes, speech signals plus noise overlay, were run through a graphic level recorder,

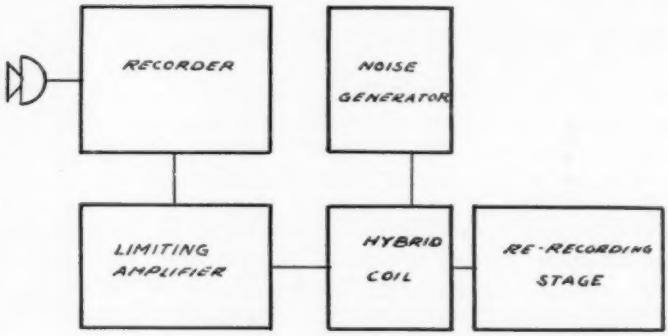


Fig. 1. Schematic Diagram of Stimulus Preparation.

Sound Apparatus Co., Model HPL. The resultant tracings verified the speech peaks as being 4 db. above the noise level. Fig. 1 shows this setup schematically.

C. Listeners—Listeners were 204 AFROTC cadets at The Ohio State University. They were divided into five groups of about 40 persons each.

D. Test Administration—All listening groups were instructed in the same manner on the mechanics of filling out the test blanks. Stimuli were then administered with an Ampex 600 tape recorder and an Ampex 620 speaker-amplifier system to listeners seated in an average-size classroom. The nearest listeners were approximately six feet from the loud-

speaker, and the playback level measured 70 db. (C scale) at a point ten feet in front and on the axis of the loud-speaker. Each listening session lasted 30 minutes, with each group of 40 evaluating the speech produced under one of the five masking conditions.

E. Analysis of Data—Intelligibility scores were computed for words and sentences. The latter were scored by percentage of key words correct, the former on percentage correctly identified. Physical measurements of intensity and duration of the original, noise-free speech signals were made and treated with analysis of variance for differences. Experimental data were also arrayed in graphic form to show trends.

III. RESULTS AND DISCUSSION.

A. Intelligibility—Fig. 2 shows the intelligibility of both words and sentences as heard in a given constant S/N ratio. To determine the extent of possible score, one of the test tapes was presented to a group of 40 listeners without any noise masking. A score of more than 98 per cent on both words and sentences was achieved, indicating almost perfect reception under excellent reception conditions. Data for listening sessions with speech produced in quiet, 70, 80, and 100 db. of speaker-masking appear in Fig. 2, scores for the 90 db. recording level being unfortunately not available, due to an administrative error in giving the tests. The regular behavior of the adjoining conditions would indicate, however, that similar results would have been obtained.

Of immediate interest is the difference in intelligibility scores between the quiet-produced and noise-produced speech. It may be seen that the first level of noise in the speaker's ears (70 db.) resulted in speech 35 per cent more intelligible in respect to words, 27 per cent more intelligible in respect to sentences. From that level on, no statistical changes in scores are noted. It is apparent, therefore, that throughout the range tested, no advantage was gained by the speakers from the heavier masking levels used during recording. The curves also indicate that during the steady-score region of 70 to 100

db. the context offered by the sentence settings resulted in about 10 per cent more intelligibility. From these results we would assume that a real and sizeable increase in the intelligibility of speech is realized by introducing some degree of noise into a speaker's ears. It may be possible that the same amount of increase might be effected with less masking noise, and it is also possible that masking levels higher than 100 db. might produce different results.

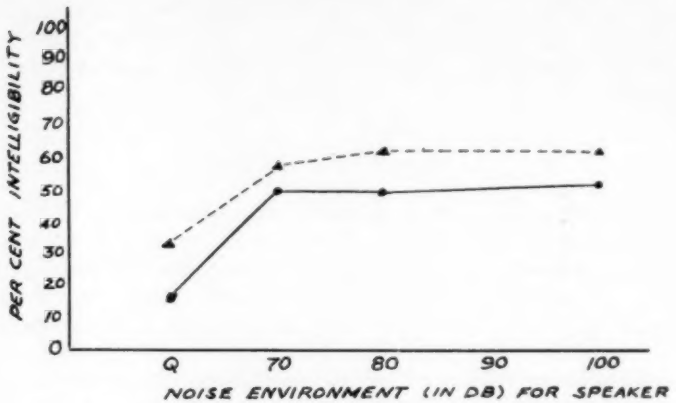


Fig. 2. Intelligibility of words and sentences spoken in various levels of noise and heard at a constant S/N ratio of zero db.

B. Physical Measures—1. Intensity Changes—Although intensity of the voice did not contribute to the intelligibility results, the original recordings could be measured to estimate the effect of speaker-masking on his production levels. The initial stimulus tapes were measured on the graphic level recorder to determine both duration and intensity. Results re .0002 dyne/cm^2 are shown in Table I.

The largest intensity increase comes, both for words and sentences, when the first level of masking is employed. After that, succeeding increments of 10 db. masking produced an average vocal increase of approximately 1 db. This estimate of 0.1 db./db. differs from a previous estimate by Korn[3] of

TABLE I.

LEVEL OF SPEECH OUTPUT UNDER CONDITIONS OF BROADBAND RANDOM NOISE MASKING AT THE SPEAKER'S EARS.

Condition	Words (db.)	Diff. (db.)	Sentences (db.)	Diff. (db.)
Quiet	65.0	.....	68.8	.....
70 db.	70.8	5.8	71.9	3.1
80 db.	71.9	1.1	72.8	0.9
90 db.	72.8	0.9	73.9	1.1
100 db.	74.1	1.3	74.9	1.0

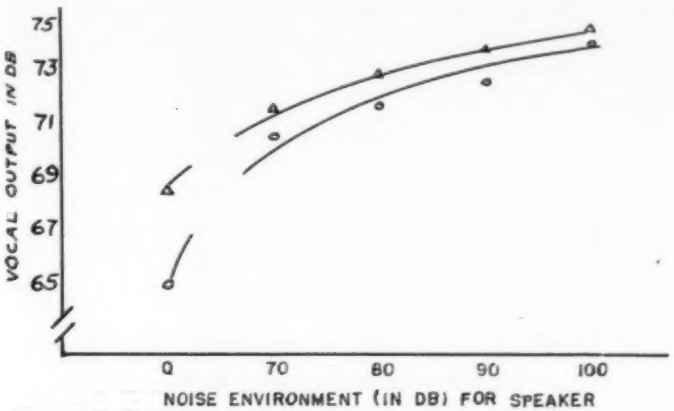


Fig. 3. Increase in vocal output with increase of masking level at the speaker's ears.

0.38 db./db. From his description of procedure it is possible that this higher figure might reflect some measurement of breath blast caused by microphone placement in his experiment. The trend of the tabled values is shown in Fig. 3. An interesting point here is that in all conditions the level for spondee was less than for sentences, both readings being taken on maximum peaks of the utterances.

2. Duration Changes—The same recording traces measured above for intensity changes yielded the comparative duration measurements tabled below:

As in the case of intensity, increased masking level resulted in a steady increase in duration, with the greatest difference

TABLE II.

DURATION (IN CENTISECONDS) OF WORDS AND PHRASES PRODUCED UNDER CONDITIONS OF BROADBAND RANDOM NOISE MASKING.

Conditions	Words	Diff.	Sentences	Diff.
Quiet	67.0	.....	170.0	.....
70 db.	77.0	10.0	197.0	27.0
80 db.	84.0	7.0	202.0	3.0
90 db.	86.0	2.0	210.0	8.0
100 db.	88.0	2.0	213.0	3.0

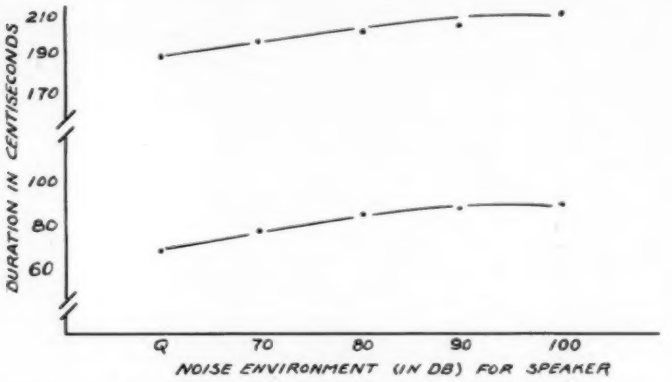


Fig. 4. Increase in duration of words and sentences with increase of masking in speaker's ears.

coming with the first onset of the masking noise. Duration functions for the two types of test material are shown in Fig. 4.

Since the intensity factor had been controlled, it cannot be held accountable for the observed increase in intelligibility score associated with noise-produced speech; and in view of the fact that duration changes from one condition to the next were so small as to be statistically insignificant, we should most logically look to other dimensions of the voice for the reasons. The answer may lie in the area of less well defined measures, such as quality and stress, or in the relationships between the sequences of the phonemes making up the speech signal. Suffice it to say, however, that the magnitude of

intelligibility change between the first and second experimental conditions might make it well worthwhile to attempt further exploration and application of it. The notion of a device for military communication would seem to be feasible for immediate development after additional tests on the range of speaker masking between quiet and 70 db. to discover minimum masking level. Further studies should also be made on the extent of speaker accommodation to the masking influence. The stable and statistically nonsignificant intelligibility scores obtaining in test materials produced under noise conditions of this study may also offer a tool to the audiologist who employs live voice presentation of tests.

IV. CONCLUSIONS.

From the evidence presented above it is concluded that:

1. There is a measurable and important increase in the intelligibility of both words and sentences produced by speakers operating with a broadband random noise mask in their headsets.

2. The intelligibility values of speech produced under 70 db. of masking were statistically equivalent to those produced under 80 and 100 db. of masking.

3. Plots of both mean duration of speech elements and vocal output show a regular increase with increase in speaker masking.

4. The range of masking between "Quiet," (estimated by curvilinear extrapolation to be approximately 54 db.) and 70 db. of masking needs further exploration to determine additional points on both the intelligibility and vocal output curves, and it is suggested that live voice tests for hearing evaluation consider the use of sufficient speaker masking to effect the vocal stability observed in this study.

5. It is recommended that the observation of increased intelligibility be put to operational test in aircraft circuits.

6. It is recommended that the aforementioned operational test, if successful, should result in the engineering of a device

for emergency use in air-ground communications for speakers in relatively quiet speaking environments.

BIBLIOGRAPHY.

1. Footnote on first page.

2. HANLEY, T. D., and STEER, M.: "Effect of Level of Distracting Noise upon Speaking Rate, Duration, and Intensity." *JSHD*, Vol. 14, pp. 363-368, Dec., 1949.

3. PICKETT, J. M., and KRYTER, K. D.: "Prediction of Speech Intelligibility in Noise." AFCRC TR 55-4, June, 1955.

4. KORN, T. S.: Effect of Psychological Feedback on Conversational Noise Reduction in Rooms. *Jour. Acoust. Soc. Amer.*, 26; No. 5:793, Sept., 1954.

A COMPARISON OF PICTURE RESPONSE AND HAND RAISING TECHNIQUES FOR PURE TONE AUDIOMETRY WITH YOUNG CHILDREN.

BRUCE M. SIEGENTHALER,
and
HARRIET KAPLAN,

The Pennsylvania State University,

University Park, Pa.

In order to teach the child with a hearing loss to utilize his hearing to his best advantage, an early educational program is necessary. This requires early detection of reduced acuity, and it becomes desirable that hearing testing techniques be developed for use with the young child.

Among the methods in current use for testing young children are noise makers, speech reception, subjective pure tone audiometry, and PGSR. Pure tone audiometrics are preferable to measurement with noise making apparatus and speech for at least part of the testing protocol, because the audiometer gives information about acuity at specific frequencies, and intensity levels are calibrated. In some quarters the PGSR test for children is not considered desirable. (Although pure tones are used, validity and reliability of testing are questioned

because of subjectivity in interpreting results and difficulties in conditioning. In addition, PGSR sometimes is considered to be unpleasant for the child and detrimental for follow-up testing and training). For these reasons subjective pure tone audiometry continues to be the method most generally preferred for testing the hearing acuity of children; however, many children will not respond to pure tone audiometry when the standard response is used (*i.e.*, when the child is expected to raise his hand when he hears the tone). The test procedure must be altered so that suitable responses can be obtained. Pure tone test procedures for children should satisfy at least the following criteria[1,2]:

1. Simple association between pure tone stimulus and response.
2. Motivate the child.
3. Take into account child's short attention span.
4. Require only a gross motor response.
5. Not require a verbal response.
6. Aid the examiner to gain rapport.
7. Produce valid and reliable results.
8. Preferably require only one examiner.
9. Allow monaural measurements.
10. Be economical.
11. Require only simple manipulation of apparatus.

Although the standard pure tone audiometry method satisfies some of these criteria, in several important aspects it may be improved upon.

Attempts to devise pure tone audiometric procedures for children have centered mostly around techniques for motivating the children to listen attentively and to respond appropriately. To accomplish these goals a number of varieties of the peep-show technique have been devised.[3,4,5] In all cases, the basic situation is that the child is rewarded for making a correct response when the tone is on, and he is not rewarded if he responds when the tone is not on. Other techniques for pure tone testing with children have been to associate objects with the audiometric tones, and to carry on pre-testing training programs to associate sound with a response.[6,7,8,9]

Although the literature on methods for testing the hearing of young children is growing, little has been done to compare the results of acuity measurements made using various subjective techniques. It was the purpose of this study to compare the standard technique (hand raising) with a picture response technique (modified peep-show) for pure tone hearing testing of pre-school children. The two response techniques were compared with respect to eight criteria, presumed to be important for evaluating a hearing test, mentioned below.

PROCEDURE.

Thirty-nine normal hearing children were used for the study. They were pupils at the nursery school of The Pennsylvania State University, and all were children of faculty members of the university, or of business men of the town. Their chronological ages ranged from three years three months to five years two months. Their mental ages, according to a Stanford-Binet test, ranged from three years five months to six years nine months. The average I.Q. of the children was 121. There were 19 males and 20 females. (Although as a group the children were of superior mental ability, this factor would not be expected to be critical to interpretation of the experimental results. Each child acted as his own control when comparing the two test methods.)

A modified peep-show apparatus was used for part of the experimental testing. Switching circuits were arranged, so that if a child heard a tone in his earphone (monaural) and pressed the top of a box on the table in front of him, the lights in the room went off and he saw an attractive colored picture of an animal projected on a screen. If he pressed the box top when there was no tone, there was no such reward. A standard pure-tone audiometer was used. The animal pictures were on 2x2 inch slides, and projected by a semi-automatic slide projector. If the child became disinterested in a picture the examiner pressed a button attached to the projector, and a new picture became available. Testing was done in a quiet room near the nursery school. The same room and audiometer were used for the standard procedure, but

the child was expected to raise his hand when he heard the tone. Children were alternated during the testing program, so that half of them had their left ears tested first and half had their right ears tested first; half were tested with the picture response technique first and half were tested by the standard hand raising technique first. It was attempted to give each child both tests in both ears for frequencies 1000, 2000, and 500 c.p.s., in the order mentioned. Approximately two weeks after the initial testing each child was given the complete battery test again.

The two test procedures were compared on the basis of the following eight criteria:

1. Three frequency average threshold.
2. Test reliability.
3. Time required to orientate child to the test.
4. Ease of orientation.
5. Number of false responses.
6. Three frequency threshold test time.
7. Per cent of three frequency tests completed.
8. Child's preference.

Not all children could be given the complete battery test. Some cooperated during one of the tests but refused the other, or occasionally only a partial test could be obtained on an ear; therefore, in the following analyses the number of cases considered does not always reach 39, and often the number is different for the various considerations.

RESULTS.

Three frequency average threshold—The average threshold for the three frequencies on a single ear of a child was computed and used as the threshold value. Table I shows the means of these threshold values for the two test methods for left ears, and for right ears on the initial test, and on the retest. The mean thresholds were compared, using the t test for related samples. Only children on whom complete tests could be obtained were included. It can be seen that in all comparisons the picture response technique gave more acute mean thresholds than did the standard response method.

Only for the right ear on the retest did the difference between means fail to reach the .10 level of statistical significance. It also will be noticed that the retest thresholds are more acute than the initial test thresholds. Picture response thresholds averaged about 3 db more acute than than response thresholds on the initial test.

Test Reliability—To measure test reliability the test-retest threshold value 0, 0-5, and more than 5 db from the initial

TABLE I.

MEAN DB THRESHOLDS AND t TESTS.

	Picture	Standard	N	t	σ_{Mp}	P
A.D. test	9.0	12.1	19	2.79	1.10	.02
A.S. test	8.6	11.3	20	1.98	1.14	.10
A.D. retest	6.8	7.4	20	.38	1.41	.70
A.S. retest	5.6	8.8	20	1.94	1.10	.10

TABLE II.

PER CENT OF EARS SHOWING DB. VARIATIONS IN THRESHOLD BETWEEN TEST AND RETEST.

	PICTURE			
c.p.s.	N	0 db	0-5 db	<5 db
500	52	35	79	21
1000	53	26	77	23
2000	53	43	81	19
Mean %	—	35	79	21
	STANDARD			
c.p.s.	N	0 db	0-5 db	<5 db
500	36	33	69	31
1000	36	50	78	22
2000	36	33	75	25
Mean %	—	39	74	26

percentages of all ears (both left and right) deviating in threshold value 0, 0-5, and more than 5 db from the initial test on the retest were computed for each frequency, and are shown in Table II. Approximately one-third of the ears had 0 db deviation for each method, and approximately three-fourths of the ears had deviations of 5 db or less from test on retest for each method. Despite the observed per cent differences between methods for individual frequencies, the differences appear to balance out, and in general both methods

appear to have similar reliabilities. (Although it is not shown in Table II, inspection of the original data indicated that in most cases the retest thresholds were more acute than the initial test thresholds. This trend was seen in Table I.)

Orientation Time—When a child entered the testing room he was made to feel comfortable and induced to sit at the table holding his switching box. After the tester demonstrated wearing the earphones it was possible to put the phone on the child, usually without difficulty. The timing of the orientation began when the earphones were on the child, and ended when the child gave three correct responses to the presence of a 1000 c.p.s. tone presented at 50 db hearing loss. Verbal instructions and demonstrations were used to teach the child the test. The mean time required to orientate the children who were given the standard response test first was compared with the mean time required to orientate the children who were given the picture response test first. (It will be recalled that in the subject group half were presented the picture response test first, and half were presented the standard response test first.) The numbers of children in the two groups for this comparison were not the same because some children could not be orientated to one or the other tests. For the 18 children orientated to the picture response test first, the mean time required for orientation was 87 seconds; the 15 children who could be given the standard response test first had a mean orientation time of 155 seconds. A t test computed between these two means gave a value of 1.80, significant between the .05 and .10 levels.

Ease of Orientation—A judgment was made by the examiner as to the ease of orientating each child to the first test he was given. The basis for this judgment was partly on the amount of time it took to orientate the child, but largely on the degree to which his interest could be aroused and held during the orientation, and general impression of his quickness to gain insight into the task required. Of the children given the standard response test first, 70 per cent (14 children) were easy to orientate, 15 per cent (three children) were difficult to orientate, and 15 per cent (three children) were impossible to orientate. For children given the picture

test first 79 per cent (15 children) were easy to orientate), 5 per cent (one child) was difficult to orientate, and 16 per cent (three children) were impossible to orientate. Although there was a slightly greater proportion of children easy to orientate to the picture test procedure, the difference between this proportion and the proportion easy to orientate with the standard response technique was not statistically significant according to a t test (t .50, P 60).

In general, those children who were easy to orientate by one technique were easy to orientate by the other technique when it was given second in the testing sequence. Where differences existed, they always were in favor of the picture test method. For example, some children given the picture test first and easy to orientate were difficult or impossible to orientate to the standard response test when it followed.

TABLE III.

MEAN TEST TIME IN SECONDS FOR A THREE FREQUENCY TEST.

	Picture	Standard	N	σ_{MD}	t	P
A.D.	246	230	14	24.42	.68	.60
A.S.	310	261	20	19.56	2.48	.05

Consistency of Response—Consistency of response was measured by noting how many times each child made responses when there was no tone present for each of the tests. The mean number of false responses made during the standard test was two; during the picture test the mean was 1.75. A t test between these mean values was not significant (t .16, P .90).

Three Frequency Threshold Test Time—Timing of the threshold testing began after completion of the orientation, *i.e.*, after the child had made three successive correct responses. Because some children could not be tested in both ears for one or both of the test procedures, test times for right and left ears were separated, and in the analysis only ears which could be tested by both methods were considered to make possible a match groups t test. Table III shows the test times and t test results. It can be seen that the

standard response method required less time than the picture response method, but the difference was significant for only the left ears. (It was the examiner's impression that the picture response test required longer time because the children enjoyed seeing the pictures so much.)

Per Cent Complete Tests Obtained—Not all children could be tested completely (three frequencies in both ears) with both response methods. Some could be tested in one ear only by a given method, and others could not be tested at all. Table IV shows the per cent of the 39 children who could not be tested in either ear, who could be tested in one ear only, and who could be tested in both ears by each of the methods. Each child was tabulated for his testing with the picture test and for his testing with the standard test. Thirty-eight

TABLE IV.

PER CENT OF THREE FREQUENCY TESTS OBTAINED.
(39 Children).

STANDARD	PICTURE No Test	One Ear	Two Ears
No Test	18	2	21
One Ear	0	10	10
Two Ears	0	0	38

per cent (15 children) could be tested completely by both methods, 10 per cent (four children) could be tested in one ear only by both methods, and 18 per cent (seven children) could not be tested by either method; however, it will be noticed that 21 per cent (eight children) could be tested in both ears with the pictures, but not at all with the standard response method, and that 10 per cent (four children) could be tested in both ears, using the pictures, but in only one ear by the standard method. Thus a total of 69 per cent (27 children) could be tested in both ears by the picture method, but only 38 per cent (15 children) could be tested in both ears by the standard response method. (In this analysis if a child could be tested for only one or two frequencies in an ear, the result was not counted as a test for that ear.)

Child's Preference—At the end of an initial testing session

for a child he was asked which test he liked better, and which test he would like to have if he were to be tested again; also, the child's parents were sent a letter requesting them to ask their child prescribed leading questions about what happened in school that day, and to note his responses. On the evening that a child was tested the parents were interviewed by telephone regarding their child's comments. The child's statements to the examiner and the reports made by the parents were transcribed and presented to five disinterested judges. The judges were asked to categorize each child's pattern of responses into the categories, "Definite preference for the standard test," "Slight preference for the standard test," "No preference," "Slight preference for the picture test," and "Definite preference for the picture test." These categories were assigned the numerical values 1 to 5 respectively, and the numerical values given each child by the five judges were totaled. If the total for a child were more than 15 he was considered to have preferred the picture test; if less than 15 the picture test, and if it were 15 he was considered to have shown no preference. By this procedure 91 per cent (30 children) showed preference for the picture response test, 3 per cent (one child) preferred the standard response test, and 6 per cent (two children) showed no preference.

DISCUSSION.

As a result of these analyses the following conclusions can be drawn regarding each of the eight criterion for comparing the two test procedures:

1. For an initial clinical test of hearing acuity in the middle frequency range the picture response technique gives more acute thresholds than does the standard hand raising response technique. The difference is about 3 db.

2. Reliability of the two test techniques appears to be about equal.

3. Orientation time for the picture response method is shorter than for the standard method. For the present sample the difference was about one minute.

4. The picture response method is slightly advantageous

with respect to ease with which children can be orientated to give correct responses.

5. There is no significant difference in the number of false responses given during administration of the two tests.

6. The picture testing procedure takes longer for the obtaining of thresholds at three frequencies. In the present study the difference was statistically significant for only the left ears of the subjects; for these ears the mean difference was about 50 seconds.

7. With respect to the success with which a test at at least three frequencies can be done, the picture response method has a clear advantage over the standard response method.

8. Children prefer the picture response test method.

When considering these eight criteria for comparing the two test techniques, the standard response method is supported only by the criterion of test time. The criteria of reliability and number of false responses demonstrated no support for either method over the other. The picture response method was supported by the other five criteria.

It is questionable whether the issue of which test is superior should be decided by the number of criteria in favor of each; rather, the relative importance of the various criteria should be considered. Although the standard response method took less time to administer after orientation, the difference was more than cancelled by the shorter orientation time for the picture method. The picture method produced slightly more acute thresholds than did the standard response method, but this difference is of little numerical or theoretical importance. Because any threshold is determined by definition, *i.e.*, hearing acuity is judged in terms of whether a given response is obtained, it is to be expected that different definitions of threshold (different responses required) would give different threshold db values. However, the picture response method was supported by the very important criteria of interest on the part of the children and per cent of children who could be tested. Gaining a child's interest, cooperation, and test thresholds are of major importance, both for clinical examination

and for further audiological work with the child. In these respects the picture response method seems to have a clear advantage over the standard response method for testing pre-school age children.

REFERENCES.

1. Morley, D.: Rationalism in Testing the Hearing of Children. *Volta Review*, 50:486-496, 1948.

2. Schwartz, A.: "Supplementary Pure Tone Audiometric Screening Test for Pre-School Children." M. S. Thesis, U. Wisc., 1952.

3. Dix, M., and Halpike, C.: The Peep Show, a New Technique for Pure Tone Audiometry in Young Children. *Brit. Med. Jour.*, 2:719-723, 1947.

4. Waldrop, W.: A Puppet Show Hearing Test. *Volta Review*, 55:488-489, 1953.

5. Ewing, A., and Ewing, I.: "The Handicap of Deafness," p. 91-93, Longmans, Green and Co., New York, 1938.

6. Bloomer, H.: A Simple Method for Testing the Hearing of Young Children. *Jour. Speech Disord.*, 7:311-312, 1942.

7. Utley, J.: Suggestive Procedures for Determining Auditory Acuity in Very Young Acoustically Handicapped Children. *E.E.N.T. Monthly*, 27:590-595, 1949.

8. Guilder, R., and Hopkins, L.: Program for Testing and Training of Auditory Function in the Small Deaf Child During Pre-School Years. *Jour. Speech and Hearing Disord.*, 16:5-10, 1951.

9. Lowell, E., et al.: Evaluation of Pure Tone Audiometry with Pre-School Age Children. *Jour. Speech and Hearing Disord.*, 21:292-303, 1956.

FURTHER CONTRIBUTIONS TO THE E.E.G. METHOD OF EVALUATING AUDITORY FUNCTION.

A. J. Derbyshire,
Harper Hospital,
and
M. McDermott,
Children's Hospital,
Detroit, Mich.

In the year 1939 the Davises first described the E.E.G. responses to sound occurring in sleeping subjects. This most accessible fact lay unused, except in animal work until 1948, when Doerfler used the E.E.G. instrument to record electro-

dermal responses. Marcus and Gibbs, in 1948 and 1951, were the first to use electroencephalic responses to determine hearing. One study was reported by Gidoll in 1952. In 1953, Pearl, et al., reported again on the use of the E.E.G. responses in sleep to evaluate function of the ear. Since our first publication in 1956, Goldstein (1956) has reported on the use of the E.E.G. in conscious states as an auditory measurement, and Simon and Emmons (1956) have used these same endpoints.

The original goal of all these workers was a clinical one concerning the many children under 4 and 5 years of age, and the many older ones in whom lack of communication is so serious a problem that standard methods do not work. With these patients it is important to be able to measure not only their functioning level, but also to dissect out where and what types of trouble may exist.

From the data obtained by reliable standard audiometry it has been possible to infer where almost any disturbance may lie in the auditory system. These inferences, however, are only an approximation. They do not tell in a detailed way how any particular part of the system is abnormal; furthermore, reliable audiometry is most difficult to obtain in the child, where accuracy is most needed.

It is during the diagnostic and planning states in the patient's early years that we need certainly to know whether the child's problem is in reception; in coding the information into nerve impulses, or in transmitting this information to the brain; or if the communication problem might also be higher up in the central nervous system involving the decoding process, the awareness, or the integration of this information with other knowledge and memories, so that synthesis into action or speech results (Hardy, 1956); or if the delayed language might be due to psychic forces, so that the child finds that only a certain level of sound or speech is compatible with a comfortable existence. It might cost him too much emotionally to pay more attention, or invest more in the area of communication by sounds.

For the evaluation of each of these components we need to

develop standardized methods that have a known reliability, and at the same time ease of accomplishment clinically.

The E.E.G. audio method in sleep, and in repose, offers procedures which put aside the psychic factors of making decisions. These electrical patterns tap the central nervous system after at least reception, coding and transmission have occurred. It is probable that the E.E.G. audio response is not at the synthesizing or motor levels. It is likely that these responses arise from a secondary pathway that splits off the main specific projection path for hearing somewhere in the brain stem or thalamus. This side path activates a diffuse system that in turn stimulates the cortex, both generally and specifically. The result is our E.E.G. response, and we believe an accompanying change in patients' sleep level. When the results of E.E.G. audio tests with known reliability are compared to standard audiometry, and to electrodermal audiometry, etc., then we can begin to establish where, to what degree and what kinds of information loss have occurred in our patient's communication channel.

Of primary importance is a careful description of the detectable responses used in E.E.G. audiometry in sleep. We began by searching for the K formations described by Davis. They were most clear and useful, but required a moderate to deep stage of sleep, and did not cover all of the various responses. They were better seen in response to clicks rather than pure tone. There were many times when a reduction of voltage or other changes were more apparent.

We find we can describe four component parts to the total response to a pure tone of two to six seconds' duration (see Fig. 1). There is, first, an on-effect with a latency of 50-500 msec. (average 250 msec.). This is most often a K formation, or a fragment of one or a spindle of 14/sec. waves. Whether or not a K formation occurs, there is, second, a reduction of voltage, particularly of the faster frequencies in the pattern with a latency of 200 to 600 msec. (average 400 msec.). This suppression may be brief or may continue throughout the 2 to 6 seconds the tone is presented. Although this is not the only kind of response at this point in time, it is by all odds the

most common and most useful one we can identify. Less often, instead of remaining suppressed, the pattern may gradually increase its voltage, rising to a level higher than that seen before stimulation. This build up shows a latency of 0.5-1.0 sec.

Third, there are frequent off-effects, of typical K formations, or brief periods of voltage reduction. When working

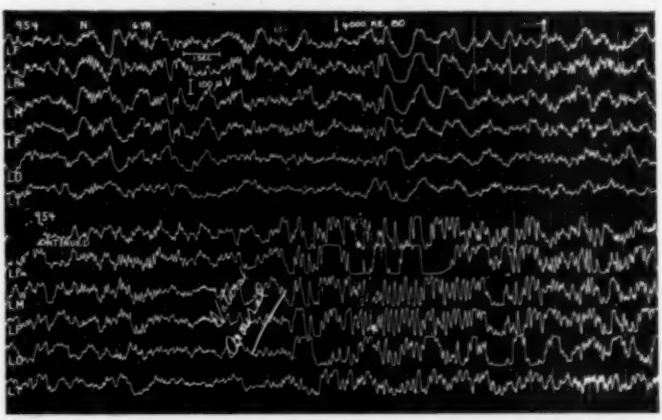


Fig. 1. This E.E.G. pattern shows three of the four possible responses to 6 seconds of pure tone. The two strips are continuous. The E.E.G. patterns are recorded between both ear lobes and LF (left frontal area), LPm (left precentral); LM (left central); LP (left parietal); LO (left occiput); LT (left mid-temporal). This is a normal hearing, six-year-old patient. Arrows mark on and off of a 4000 c.p.s. tone at 80 db. to the right ear. A K formation begins after a latency of 0.8 seconds (on effect). There is a suppression of fast rhythms, in frontal areas from 1.4 sec. to 3.6 sec. (continuous effect) although delta (1-2/sec.) persist. There is no off effect other than the return of 11/sec. spindling with the cessation of tone. Seventeen seconds after the stimulus starts there is a dramatic change of pattern signaling an arousal. The child woke up, stirred and fell asleep again. The words "natural arousal" written on the record were our notes, meaning that no other stimulus was identifiable in the 17 sec. interval.

near threshold these are often stronger than the on-effect. Just as the conscious patient often misses the onset, but detects the turning off, of the presented tone when at or near threshold.

Fourth, there is a late effect. Any time within about 25 seconds of turning on a tone of 5 seconds' duration there may be a relatively sudden change in the entire pattern of the

E.E.G., signifying a different level of sleep. The subject may awaken at such a time, or the change may be only transient. Rarely the E.E.G. may show a deeper level of sleep.

These responses do not all occur with every stimulus. They may each vary in amplitude, duration and complexity; in fact, if one stimulus is repeated rhythmically, as Chafetz and Cadilhac (1954) have shown, the response tends to vary in complexity, amplitude, and ease of detection. It comes and goes over periods of seconds to minutes. Peculiarly, not all cortical areas wax and wane synchronously, so that the most clearly visible response may shift from area to area. The form of the E.E.G. responses show little evidence of relationship to intensity of stimulus as we test for them. This relationship of form of response to intensity could well be masked by our method of study. It is clear that since our latencies vary from the shortest of 50-70 msec. to well over 2.0 seconds we are dealing with a complex system. In fact, because the response is, *1.* independent of age (down to 2 months); *2.* has such a long and variable latency; *3.* is most prominent in the fronto-central areas, we have proposed the idea that the response is evoked in the cortex, not by the primary projection path to the temporal lobe, but by a side path to the reticular formation of the midbrain or thalamus. The question we cannot answer is whether or not every E.E.G. change we identify could be heard by the patient if he were conscious and attending. There is the possibility of sound arriving through the ear into the central nervous system that the patient could not consciously detect or hear.

Accepting these E.E.G. changes in sleep as responses to pure tone, the next question is what is the threshold of this E.E.G. response, and what relations does it have to conscious hearing. We define as threshold the intensity level producing 50 per cent positive response.

The comparison of E.E.G. threshold with standard audiometric thresholds in a group of children in a deaf school showed a deviation of ±18 db. Recently we determined the variation between repeat E.E.G. audiometric tests in sleep.

These repeats were done because we were dissatisfied with the results on the first test for one reason or another. Here again the variation was ±18 db. We believe these express the worst possible errors in the method, and before giving a final statement we intend to describe more fully the nature of the most reliable response and the rules for its detection. We believe we can do better, because the consistency within one test is much better than the figure of ±18 db. When there is deviation in our comparisons it is consistent and applies to all points in that test.

Our first attempts to solve the reasons for these differences is the work we are reporting here. We will describe these E.E.G. responses as a function of intensity over a wide intensity range. It should be emphasized that thresholds, regardless of their usefulness, are but a series of points along one edge of a vast field of sensory experience. A threshold is a function tested at its limits, where it is most likely to be non-linear. At threshold the mechanism may function on some other principles than it would at the levels used for ordinary communication. Finally a threshold is where error is likely to be greatest.

There are two aspects of E.E.G. responses which we can measure easily as functions of intensity: one is the latency of the response; the other is percentage of positive responses identified at each intensity level, and these can be studied in repose and sleep states in the same patient. In fact, on several occasions it has been possible to record on the 8 channel E.E.G. machine a signal of the tone presented, a signal activated by the patient's closing a micro-switch held in his hand, the potentials of the muscles used for that action, the E.E.G., the E.D.G. and either a pneumograph of the chest or a plethysmograph of a finger (see Fig. 2). This record may start in repose, and after obtaining the function relating intensity to response, the patient may fall asleep, and we can repeat the function in sleep. The percentage of positive responses as a function of intensity can then be compared in repose and sleep.

It is often important to plot the latency in seconds as a

function of intensity in decibels simultaneously with the per cent response curve. Latency approaches infinite duration at some minimal intensity. With increasing intensities the latency shortens until it approaches a minimal constant value. This curve is so close to an hyperbola that if it is plotted as the reciprocal of the latency against intensity in decibels a straight line results. Near threshold the points tend to

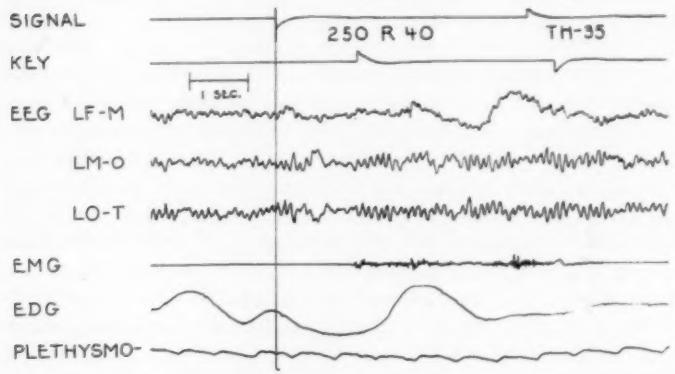


Fig. 2. This is a strip of polygraphic recording from a patient in repose, showing the responses to a tone of 250 c.p.s. to his right ear at 40 db., or 5 db. above his standard audiometric threshold. The latency of closure of key is 1.4 seconds. The latency of suppression of Alpha is 0.7 seconds. The muscle potentials appear at 1.3 seconds. The electrodermal response occurs at 1.9 seconds, while the plethysmograph change occurs at almost 4 seconds. With cessation of tone there is opening of key in 0.5 seconds, and Alpha suppression in 1.3 seconds, while the muscle potentials end in 0.35 seconds.

deviate from the straight line function and may follow some different law, or it may be that since data is difficult to obtain at those low intensity levels in any quantity, inaccuracy makes results unreliable.

By examining the functions of both latency and per cent response, it is more apparent when an error in reading has occurred. In such a case the points at one sensation level on both curves deviate widely from the expected or smooth function. By re-examining the raw data for these points one can learn about the errors in judgment likely to be made. There is often wide variation around each point, but the consistency

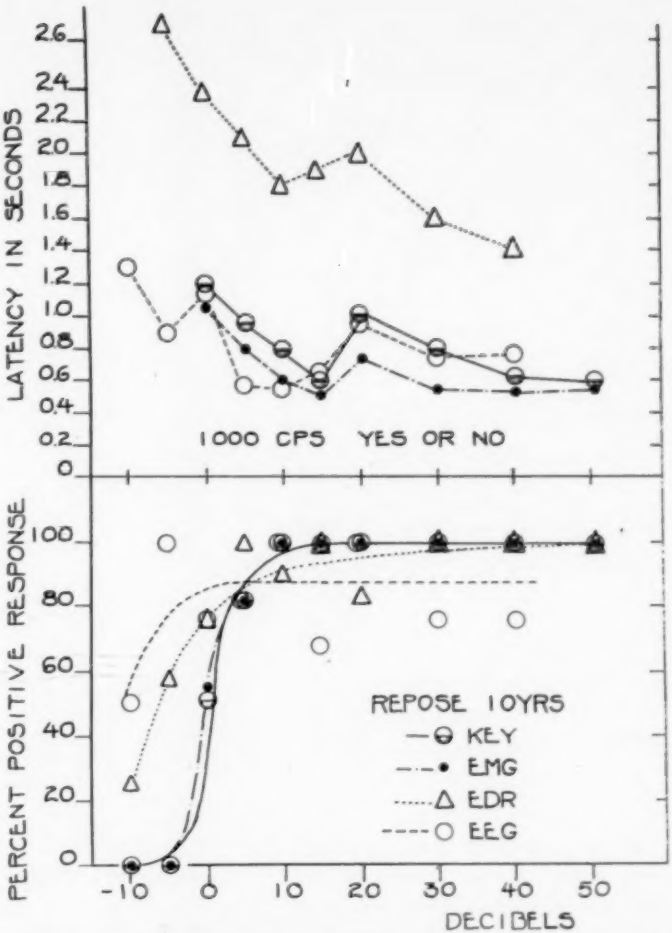


Fig. 3. These are the plots of the latencies in the upper graph and the percentages of positive responses in the lower graph for 4 of the phenomena recorded polygraphically. This data is obtained from the same patient in repose as shown in Fig. 2, except that this is for 1000 c.p.s. to right ear. Note that the latency of Alpha suppression is shorter than latency of muscle potentials till the level of 15 db. is reached. Above 15 db. the muscle has the shorter latency.

Key—Closure of micro-switch held in patient's hand; E.M.G.—electromyal responses from the muscle group for flexing palm and fingers of hand holding key; E.D.R.—Electrodermal responses as potential changes between palm and back of hand; E.E.G.—electroencephalic responses from leads covering frontal motor and occipital zones. Decibels in this paper are all relative to 0 db. as the normal human threshold for that frequency.

with which the averages yield the same shape of curve convinces us of the presence of the two functions at this low intensity edge of the auditory field.

The comparison of these functions with different kinds of communication problems, and the comparison of repose and sleep, bring out information about our original problem of the differences between standard and E.E.G. audiometry.

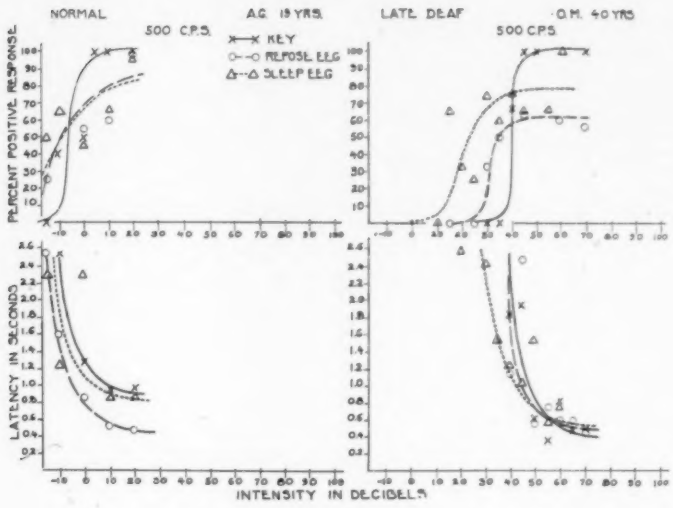


Fig. 4-A.

An illustration of these functions as displayed by the various end points of response in the waking normal subject are illustrated in Fig. 3.

The unconsciously controlled components *i.e.*, E.E.G. and E.D.G. tend to show a higher percentage of positive responses and shorter latencies than those of the E.M.G. and the key at low intensity levels. These responses may reflect the presence in the central nervous system of the information upon which the closing of the key is made. It is of interest to see the presence of muscle tension without closure of key

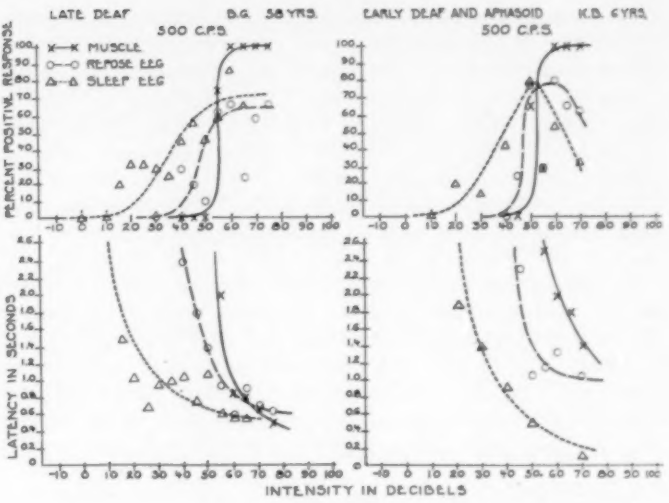


Fig. 4-B.

Fig. 4, A-B. This figure presents the plots of latencies in the lower curves and of the percentages of positive responses in upper curves. The three individual curves represent the E.E.G. responses in sleep, the suppression of Alpha in the E.E.G. of repose and either the muscle potentials or closure of key in repose. The four patients are A. G., a normal; O. M., middle aged patient with otosclerosis of some eight years duration; B. G., an elderly person with deafness occurring late in life on the side being tested, and K. B., a young child who clinically has evidenced both deafness and language retardation since the age of speech development. These functions were all studied at 500 c.p.s.

The reversal of latencies between Alpha suppression and key is not seen in all patients in the range studied. The functions of the unconscious responses (E.E.G.) arrive at a maximal percentage and minimal latencies about the intensity level, at which the conscious response is making its most rapid changes.

On patient K. B., is indicated a phenomenon which is not rare. The per cent of positive E.E.G. responses falls off at intensities higher than those which achieve the maximum. This suggests inhibitory phenomena. (Hardy, 1957).

when working at minimal intensities. It is most obvious that the subject hears, even though he decides not to respond.

At minimal levels of intensity the suppression of Alpha appears as the first response in time and the muscle potentials come later in time. About 20 db. above this level there is a trend for reversal. The muscle potentials appear first and the Alpha suppression follows. We assume that the Alpha activity is suppressed by an increase in attention. In the light of this concept, then, the Alpha suppression would be

much more likely at low intensity levels, where the signal is hardly distinguishable from the background noise, and an increase in attention is required for detection of the sound. When the tone is 20 db. stronger, tnen no change in attentiveness is required for detection, so the Alpha may not suppress; however, we often now find the Alpha suppressed after the appearance of the muscle potentials for closing the key. It is probably the increase in alertness resulting from the movement which produces this Alpha suppression.

In Fig. 4 are shown four pairs of these same curves of latency and per cent positive response related to intensity for four persons (one normal and one deaf from otosclerosis, one deaf late in life and one child with both aphasia and deafness). In each graph are presented simultaneously the three curves found for the key or muscle response, the suppression of Alpha in repose and the E.E.G. response in sleep.

The relationships between these three measurements indicates that the consciously determined response of muscle or key has a shift from 0 to 100 per cent positive response over only a narrow intensity range. The E.E.G. response in repose has a less steep slope, and rises over a broader intensity range, while the E.E.G. response in sleep has a still more gradual rise or slope. The trend, however, is for the E.E.G. functions to arrive at their maximum per cent at the same intensity level at which the key or muscle response suddenly rises to its maximum.

The function of the muscle, or key response, expresses the probability of occurrence of a conscious decision. This decision is determined by many factors, which are at present being investigated by W. Tanner (1956). Over and above the signal to noise ratio these factors include the various values and costs of correct and erroneous judgments within the situation. The decision also depends upon the clarity in the patient's memory of the tone for which he is listening, as well as his ability to listen.

Since the E.E.G. functions are a correlate of alertness and arousal, it is significant that these reach their maximum at the time when the key response makes its rapid rise to maxi-

mum. It appears that the conscious audiometric threshold is the level at which near maximal alertness has been achieved by the tone.

J. J. Groen (1957) has suggested that these curves represent probability functions, in which the coincidental presence of the first two (E.E.G. response in sleep and in repose) might represent activities in the nervous system which cause the third (response of the key). It seems as though each function might represent a nucleus on the way to the cortical surface.

The response of the key would reflect cortical activity. The Alpha suppression in repose would reflect the action of some nucleus below the cortical level, perhaps in the thalamus. The E.E.G. responses in sleep would reflect a still deeper nucleus or center perhaps in midbrain.

SUMMARY.

The characteristics of responses to tone in the E.E.G. in sleep are reviewed. On, continuous, off and late effects are described.

The latency of the first identifiable response, and the per cent positive responses are studied as functions of intensity.

Three functions are compared.

1. In sleep those described above (an unconscious response).

2. In repose the Alpha suppression (an unconscious response).

3. In repose the closure of a key (a consciously determined decision).

The function of per cent positive responses in sleep has the most gradual slope, while the closure of the key has the steepest slope.

The latency at minimal intensities is shortest for the E.E.G. response in sleep, while it is longest for closure of key. The difference is lost, or the reverse is true at the higher intensity levels.

The E.E.G. response in sleep and the Alpha suppression in repose are interpreted as signs of alerting mechanisms of the reticular formation. These functions reach their maximum at the same intensity level at which the per cent response for the closure of the key reaches its maximum. This suggests that maximal alertness is one element in determining the standard audiometric threshold.

REFERENCES.

CHAFETZ, M. E., and CADILHAC, J.: A New Procedure for a Study of Barbiturate Effect and Evoked Potentials in the E.E.G. *E.E.G. Clin. Neurophysiol.*, 6:462-472, 1954.

DAVIS, H.; DAVIS, P. A.; LOOMIS, A. L.; HARVEY, E. N., and HOBART, G. A.: Electrical Reactions of Human Brain to Auditory Stimulation. *Jour. Neurophysiol.*, 2:200-514, 1939.

DAVIS, P. A.: Effects of Acoustic Stimuli on the Waking Human Brain. *Jour. Neurophysiol.*, 2:494-499, 1939.

DOERFLER, L. G.: Neurophysiological Clues to Auditory Acuity. *Jour. Speech, Hear. Dis.*, 13:227-232, 1948.

GIDOLL, S. H.: Quantitative Determination of Hearing to Audiometric Frequencies in the Electroencephalogram; Preliminary Report. *A.M.A. Arch. Otolaryngol.*, 55:597-601, 1952.

GOLDSTEIN, R., and CHARAN, K.: *Jour. Speech and Hear. Dis.*, 1957. In Press.

GROEN, J. J.: University of Utrecht, Netherlands. Personal Communication, 1957.

HARDY, WM.: Problems of Audition. Perception and Understanding: Differentation and Need. *Volta Revief*, 58:289-300+, Sept., 1956.

HARDY, WM.: Personal Communication, 1957.

MARCUS, R. E.; GIBBS, E. L., and GIBBS, F. A.: Electroencephalography in Diagnosis of Hearing Loss in the Very Young Child. *Dis. Nerv. Syst.*, 10:170-173, 1949.

MARCUS, R. E.: Hearing and Speech Problems in Children; Observations and Use of Electroencephalography. *A.M.A. Arch. Otolaryngol.*, 53:134-146, 1951.

PERL, E. R.; GALAMBOS, R., and GLORIG, A.: The Estimation of Hearing Threshold by Electroencephalography. *E.E.G. Clin. Neurophysiol.*, 5:501-512, 1953.

SIMON, CHARLES W., and EMMONS, WM.: E.E.G., Consciousness and Sleep. *Science*, 124:1066-1069, 1956.

TANNER, JR.; WILSON, P.; SWATS, JOHN A., and GREEN, DAVID M.: Technical Report No. 30. General Properties of the Hearing Mechanism. Electronic Defense Group, University of Mich., 1956.

SESSION B.

May 16, 1957.

BEHAVIOR OF THE NEUROGLIA OF THE ACOUSTIC NUCLEI, SUB-CORTICAL CENTERS AND AUDITORY CORTEX, OF CATS INTOXICATED WITH DIHYDRO-STREPTOMYCIN.

GERALDO DE SA,

University of Recife, Brazil.

Neurotoxic phenomena caused by the use of antibiotics, are well known; however, the specific toxicity of streptomycin for the vestibular system, and of dihydro-streptomycin for the cochlear system, is interesting. Literature on the subject is wide, and we should like to remember the papers of Fowler and Seligman[1]; Carr, Brown, Hodgson, and Heilman[2]; Shane and Laurie[3]; Glorig[4]; Heck, Lynch and Graves[5]; Caussé[6]; etc.

Considering the neurotoxic phenomena doubtless induced hematically, we tried to verify the behavior of the nourishing element of the nerve tissues; the neuroglia, in the several nuclei of the cochlear pathway; the sub-cortical centers and in the auditory cortex. No reference was found to this important complement of the nervous tissue in more than 40 bibliographic sources. Taking advantage of the known specific toxicity of dihydro-streptomycin for the cochlear system, we endeavored to bring some light to the confused cochlear pathway and cortical projection on an experimental animal. Unable to use the monkey, we chose the cat, whose nervous system has been thoroughly investigated.

ANATOMIC AND PHYSIOLOGIC DATA.

Stimuli received by the organ of Corti pass the dorsal and ventral cochlear nuclei; from there they go through the

superior olive, trapezoid body nucleus and the opposite lateral lemniscus nucleus to the inferior colliculus (posterior quadrigeminal tubercle) and to the medial geniculate body, which also receives stimuli from the inferior colliculus and from fibers of the opposite nerve, through the so-called Gudden commissure (Castaldi, 1920[7]). It is evident, that the inferior colliculus has a double action: *a*. Interposed center in the superior pathway; *b*. Reflex center. Cochlear reflex movements of the eyes and neck are so determined. It is, therefore, established that the true acoustic sub-cortical center is on the medial geniculate body, where the acoustic radiation that goes to the cortex originates (Becari, 1943[8]).

The center of the acoustic projection is little known in experimental animals. In the human, it is localized above the the superior temporal gyrus, (Pfeifer, 1920[9]), and constituted by separated striae organized in different physiologic zones, placed according to Campbell[10] and Penfield[11] on the areas 22, 41, 42 of Brodman's chart.

Walker[12] described the acoustic projection on the monkey. Very little is known about the other vertebrates, including the cat, besides Cajal's (1911) demonstration of elements that were similar to the acoustic cells of man.[13] Functional localizations were well observed on the carnivora, but the uncertainty about the acoustic area remained, due to the poorly defined relations of the temporal sulci. The disagreement among writers reached its peak regarding the identification of the so-called ecto-sylvian sulcus.

Fig. 1 shows the lateral views of the brain of the cat, according to Dusser de Barenne[14] and of the cercoleptes caudivolvulus, according to Brodman.[15] The morphologic difference of the topographical arrangements of the sulci of the temporal lobe, mainly the ecto-sylvian sulcus, is clear. In the cat, the sulcus is called pseudo-sylvian and located in front, of the anterior ecto-sylvian sulcus and behind the posterior ecto-sylvian sulcus.

The well-known neuroglia was mainly studied by the Spanish School, Cajal, 1911, 1920[13]; Rio Hortega, 1921, 1942[16]; De Castro, 1942[17]; by the Italian School, Golgi, 1903[18]; Cer-

Fig. 1.

letti, 1915[19]; Loreti, 1938[20]; Bairati, 1950[21]; and Contu, 1953[22]; by the Anglo-American School, Penfield, 1924[23]; and Robertson, 1900,[24] and by the German School, Nissl, 1904, and Bauer, 1924.[25]

Bairati and Contu established the glial architecture of the nervous centers, the former in the amphibia and avis, and the latter in the reptilia and mammalia. These authors proposed the following classification:

Type 1. Architecture of the white substance: gliocytes arranged around the myelin sheath, bound among themselves and with the vascular net, by other glial elements, forming a connective apparatus.

Type 2. Architecture of the brain stem nuclei: gliocytes forming perineuronal sheaths arranged as a connective apparatus with the blood vessels.

Type 3. Architecture of the supra axial centers: gliocytes articulated with the glial processes and with the vessels.

Studying the medulla oblongata of the cat, Contu found glial architecture of the second type, formed by fibrous elements with numerous satellite oligodendrogliocytes. In the inferior colliculus the same aspect was seen: innumerable oligodendrogliocytes with regular cuboid body, thin, smooth, hairlike processes, scarcely ramified. In the young animals, however, the architecture is constituted by protoplasmatic elements; also scarcely ramified, epithelioid elements and small oligodendrogliocytes. The former aspect, according to the author, would be the result of age.

Some authors reported glial intoxication. Among them, Sancez Ibañez and Perez[26] in 1935 performed guanidinic intoxication and observed gliosis and granular degeneration of the processes' cytoplasma.

Stigliani[27] in 1940, demonstrated in the toxi-infections a great amount of neuroglia, and writes about glial hyperplasia without any statistical proof, as reported before by Lucaro[28] in 1907, even though affirming that mitosis and amitosis were exceptional on the neuroglia.

MATERIAL AND METHODS.

Five cats, two male and three female, between eight and 18 months old, were used. Meat and milk were their diet. One of the cats was used as a control (see chart). The

animals had their weight and cochlear reflexes controlled every two days. The animals were sacrificed by perfusion of the Del Rio-Hortega liquid: Distilled water, 240 cc.; Formalin, 60 cc., Ammonium bromide, 9 gr., pH = 5.

The fixation was controlled daily, and the solution changed every time it became neutral. After 15 days of fixation the specimens were frozen-sectioned and submitted to impregnation for 30 days.

CHART I
DI-HYDRO-STREPTOMYCIN INTOXICATION

DAY	CAT Nº1 CONTROL		CAT Nº3			CAT Nº5			CAT Nº6			CAT Nº8		
	—	WEIGHT IN GRAMS	ACOUSTIC REFLEXES	AS STREET GIVING 90 DB IN THE EAR	WEIGHT IN GRAMS	ACOUSTIC REFLEXES	AS STREET WEIGHT GIVING 90 DB IN THE EAR	WEIGHT IN GRAMS	ACOUSTIC REFLEXES	AS STREET WEIGHT GIVING 90 DB IN THE EAR	WEIGHT IN GRAMS	ACOUSTIC REFLEXES	AS STREET WEIGHT GIVING 90 DB IN THE EAR	WEIGHT IN GRAMS	ACOUSTIC REFLEXES
5-23-56		1330	PRESENT	0,10	1900	PRESENT	0,10	1750	PRESENT	0,10	1250	PRESENT	0,10	1700	PRESENT
5-24-56		"		0,10		"	0,10		"	0,10		"	0,10		"
5-25-56		1350	"	0,10	1683	"	0,10	1750	"	0,10	1280	"	0,10	1730	"
5-26-56			"	0,10		"	0,10		"	0,10		MODERATE	0,10		"
5-27-56			"	0,10		MODERATE	0,10		"	0,10		"	0,10		"
5-28-56		1428	"	0,10	1795	"	0,10	1820	MODERATE	0,10	1323	"	0,10	1765	MODERATE
5-29-56			"	0,10		"	0,10		"	0,10		"	0,10		"
5-30-56		1450	"	0,10	1820	"	0,10	1870	"	0,10	1400	"	0,10	1825	"
5-31-56			"	0,10		"	0,10		"	0,10		FAINT	0,10		"
6-1-56		1500	"	0,10	1860	FAINT	0,10	1900	"	0,10	1430	"	0,10	1950	FAINT
6-2-56			"	0,10		"	0,10		"	0,10		"	0,10		"
6-3-56			"	0,10		ABSENT	0,10		FAINT	0,10		"	0,10		"
6-4-56			"	0,10		"	0,10		"	0,10		"	0,10		"
6-5-56			"	0,10		"	0,10		"	0,10		ABSENT	0,10		"
6-6-56			"	0,10		"	0,10		"	0,10		"	0,10		"
6-7-56			"	0,10		BROWNESS	0,10		"	0,10		"	0,10		ABSENT
6-8-56		1450	"	0,10	1650	"	0,20	1750	"	0,20	1350	"	0,20	1650	"
6-9-56			"	SACRIFICED,			0,20		ABSENT	0,20		"	0,20		"
6-10-56			"				0,20		"	0,20		BROWNESS	0,20		"
6-11-56			"				0,20		BROWNESS	0,20		"	0,20		"
6-12-56			"				0,20		"	SACRIFICED			0,20		BROWNESS
6-13-56			"				SACRIFICED						0,20		"
6-14-56		SACRIFICED											SACRIFICED		

The Del Rio-Hortega method as used in the Anatomy Laboratory of the University of Recife includes the following rules:

1. After life fixation by perfusion, excision of the nervous system with dissection of the pons, medial geniculate body, brachium of the inferior colliculus, inferior colliculus, and cortex, corresponding to the gyri in front, and behind to the pseudo-sylvian sulcus. Specimens obtained were submitted to fixation for 15 days in the Rio-Hortega liquid.

2. Frozen sections, 15 to 25 micra thick.

3. Re-fixation in the Rio-Hortega for 15 days.

4. Washings for one hour in three distilled waters.

5. Sections submitted to Hortega's silver, to which was added two to four drops of piridin, for one hour at ambiental temperature, and for 10 to 20 minutes at 50° C.

6. Washing for 30 minutes in distilled water.

7. Reduction in formaldehyde solution by 5 per cent.

8. Washing for one hour in distilled water.

9. Turning over of the specimens in a solution of 2 per cent of gold chloride.

10. Fast washing in distilled water.

11. Reduction for five minutes in a 5 per cent solution of sodium hyposulfite.

12. Washing for one hour and alcoholic dehydration followed by mounting of the specimens in Canada balsam.

Some of the specimens have also been submitted to the Mallory and Iron Hematoxilin methods.

EXPERIMENTAL FINDINGS.

Sections of the pons, (cochlear nuclei, superior olive, and trapezoid body nucleus), mesencephalon, (inferior colliculus), metathalamus, (medial geniculate body) and cortex, (gyrus between the pseudo-sylvian sulcus and the posterior ecto-sylvian sulcus), were studied.

Pons.

Control Cat—Glial architecture of the second type was found on the cochlear nuclei, superior olive and trapezoid body. Perineuronal sheath formed by fibrous astrocytes and satellite oligodendrogliocytes nicely developing connective apparatus and connections with the blood vessels (see Figs. 2, 3).

Intoxicated Cats—Only the nuclei of the gliocytes appear; many with abundant atypical mitosis. The glial processes are

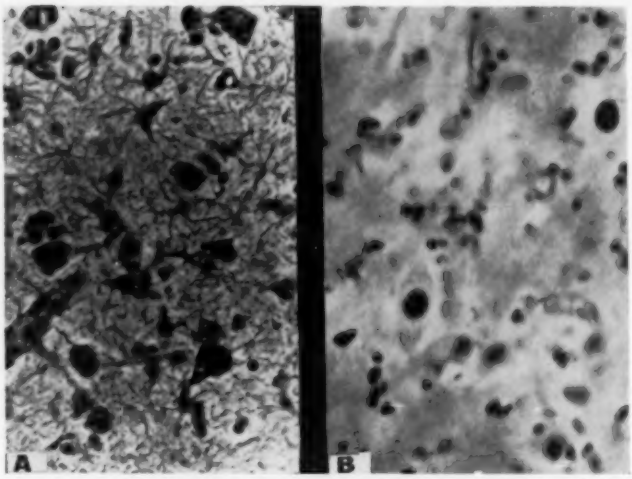


Fig. 2.
A—Section of cochlear ventral nucleus of control cat—500X.
B—Section of cochlear ventral nucleus of intoxicated cat—500X.
(Del Rio-Hortega Method)

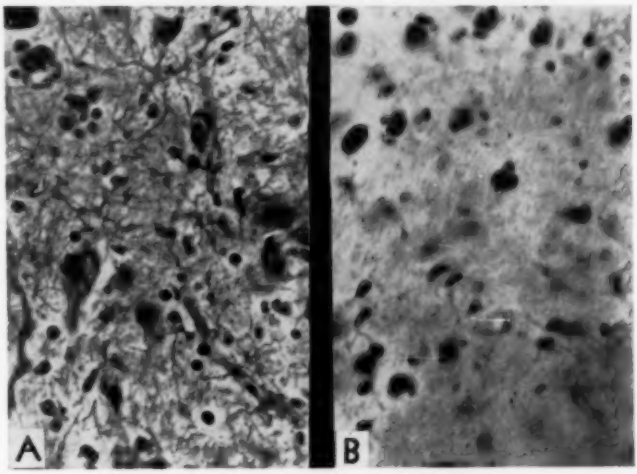


Fig. 3.
A—Section of trapezoid body of control cat—500X.
B—Section of trapezoid body of intoxicated cat—500X.
(Del Rio-Hortega Method)

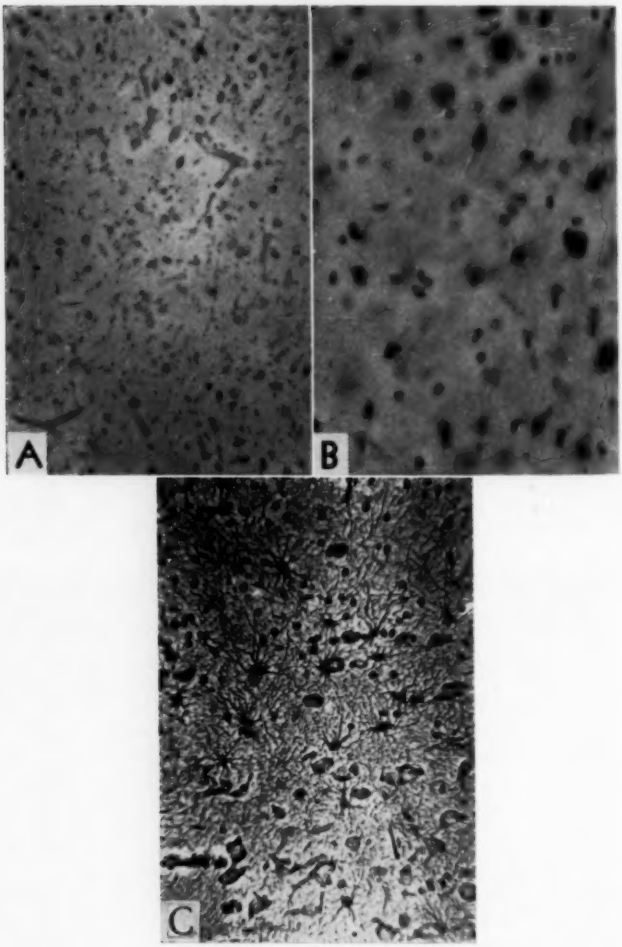


Fig. 4.

A—Section of inferior colliculus of intoxicated cat—80X.
B—Section of inferior colliculus of intoxicated cat—500X.
C—Section of inferior colliculus of control cat—145X.
(Del Rio-Hortega Method)

not demonstrable, and only a few oligodendrogliocytes present thin processes, almost unimpregnated; mitosis more frequent on the gliocytes of the connective apparatus.

Mesencephalon.

Control Cat—Typical second type architecture on the inferior colliculus. Big fibrous astrocytes with many ramifica-

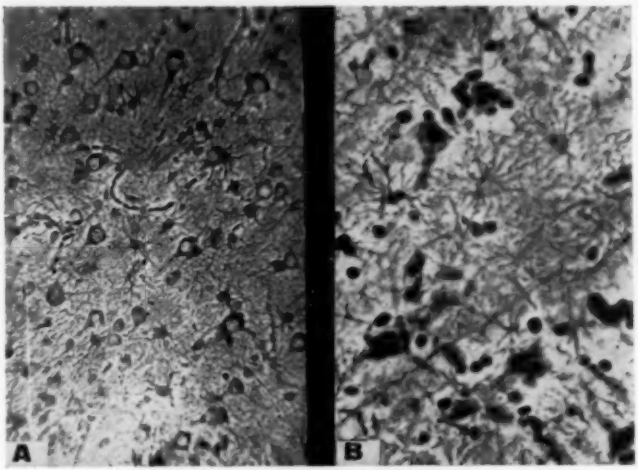


Fig. 5.

A—Section of geniculate body of control cat—145X.
B—Section of geniculate body of control cat—500X.
(Del Rio-Hortega Method)

tions. Many oligodendrogliocytes with thin and smooth processes with little protoplasmatic elements (see Fig. 4).

Intoxicated Cats—A very different aspect; atypical mitosis and only in very few elements the processes appeared slightly impregnated; most of the processes are non-identifiable.

Metathalamus.

Control Cat—Glial architecture of the geniculate body of second type, characterized by the presence of few fibrous astrocytes; many protoplasmatic processes and oligodendro-

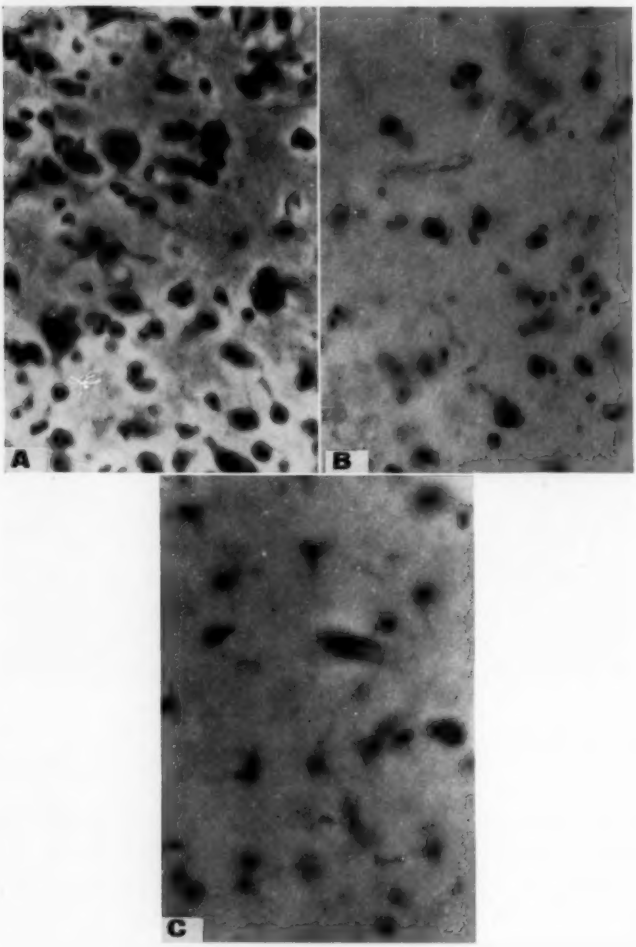


Fig. 6.

A—Section of central area of geniculate body of intoxicated cat—500X.
B and C—Section of marginal area of geniculate body of intoxicated cat—500X. (Del Rio-Hortega Method)

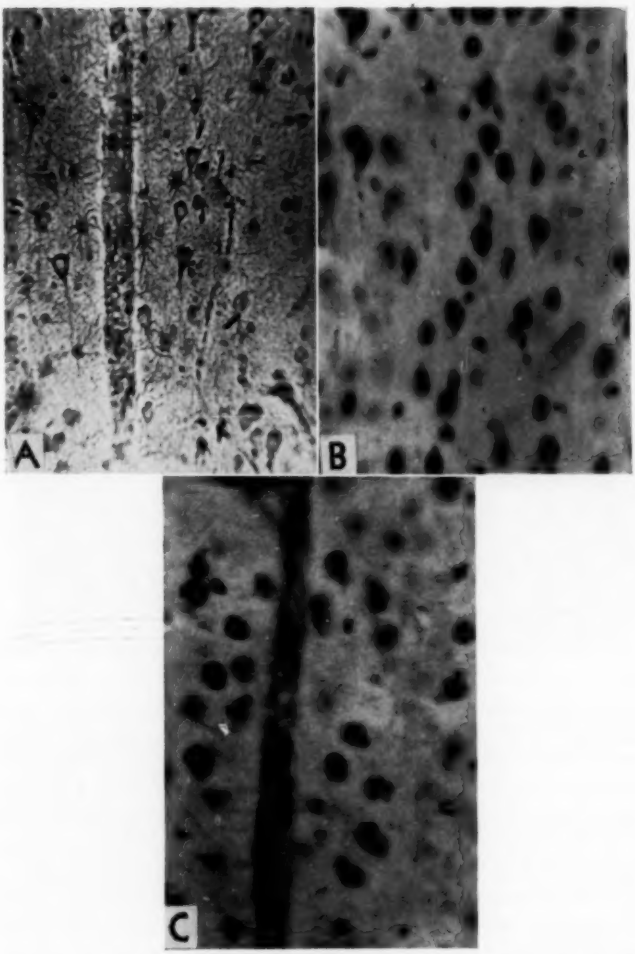


Fig. 7.

A—Section of the gyrus behind the pseudo-sylvian sulcus of control cat—145X.

B and C—Section of the gyrus behind the pseudo-sylvian sulcus of intoxicated cat—500X. (Del Rio-Hortega Method)

cytes with small processes; several gliocytes without any processes. Transitional elements were found here between protoplasmatic and fibrous processes (see Figs. 5, 6).

Intoxicated Cats—Many atypical mitosis of the gliocytes; very few impregnated processes could be found.

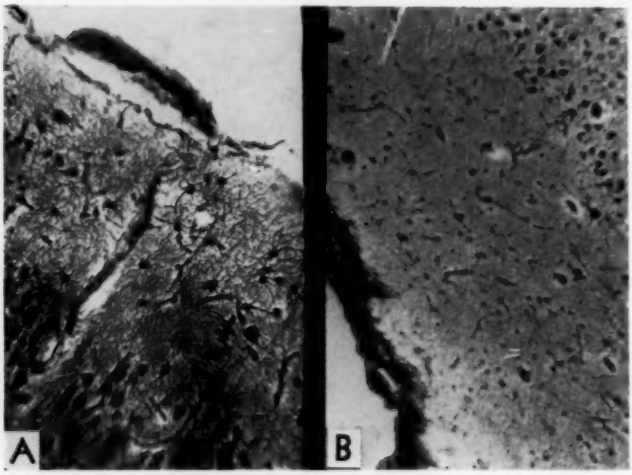


Fig. 8.

A—Section of molecular stratum of the gyrus behind the pseudo-sylvian sulcus of control cat—145X.

B—Section of molecular stratum of the gyrus behind the pseudo-sylvian sulcus of intoxicated cat—80X. (Del Rio-Hortega Method)

Auditory Cortex.

Control Cat—Glial architecture of the third type. Fibrous and protoplasmatic astrocytes; many oligodendrogliocytes with cuboid body and smooth and hairlike processes (see Figs. 7, 8).

Intoxicated Cats—Glial architecture too evident in the gyrus behind the posterior ecto-sylvian sulcus seeming a gliosis, while in the gyrus behind the pseudo-sylvian sulcus the gliocytes show no processes and some mitosis. Processes could

be detected only on the molecular stratum, even though not so evident as in the control cat.

Spinal Cord, Brain Stem and Areas of the Cortex.

Control Cat—Normal glial architectures.

Intoxicated Cats—The spinal cord shows normal glial architecture; less evident glial architecture was found in the boundaries of the pons, in the brain stem as well as in other areas of the pons themselves, as much in the white substance as in the gray; sections of the frontal, parietal and occipital cortex showed no alteration of the glial structures.

CONSIDERATIONS AND CONCLUSIONS.

Objections could be made to this experiment on the basis of the known lability of the glial structures under silver impregnation. Cerletti[19] in 1915 denied the existence of the glial processes. That opinion was, however, due to the difficulty of impregnation of glial structures permitting the appearance of technical artifacts. Different aspects of neuroglia observed under similar conditions have been reported by Cardona,[29] and Stigliani[27] assumed that the general conditions of the material would rule the glial demonstrability. Based on this assumption he explained the phenomenon of glial phanerosis, which would not be hyperplasia, but only better identification due to self and surrounding conditions. Doubtless this phenomenon is more evident in the cortex, and we found it in the gyrus behind the posterior ecto-sylvian sulcus giving the impression of gliosis. On the other hand we found that in the gyrus behind the pseudo-sylvian sulcus the glial structure disappeared, leaving only the cellular bodies, many of which presented mitosis. It seems evident that areas so closely placed and so deeply differentiated must be under different metabolic conditions. This must also be the reason for the various observations of the neuroglia.

We cannot say which is the true toxic action of the streptomycin over the neuroglia in the observed areas: whether the alterations are definitive or transitory, or whether this toxicity acts on the neuroglia alone; but we can affirm the fact that this toxic action reaches the feeding elements

of the nerve cells, inducing glial mitosis with lack of impregnation of the glial processes.

Our observations need further histological and cytochemical basis. It is our intention to repeat the experiment and study the material with a polarizer microscope to observe eventual modifications of the refringency of the processes. This, we understand, would give a better answer as to the destiny of the processes. We also think it necessary to verify the number of glial nuclei statistically, and so prove the existence of hyperplasia; also, we wonder whether the processes would remain impregnated if submitted to more intensive fixation.

With the data obtained in our experiment we can, however, conclude that the dihydro-streptomycin exerts toxic action on the centers of the cochlear pathway, as shown by atypical glial mitosis and disappearance of glial structures, well demonstrated in specimens taken from a control animal, treated with the same technical approach; furthermore, the presence of mitosis and the absence of glial processes in one area of the gyrus behind the pseudo-sylvian sulcus, permits us to assume that this is the site of central acoustic radiation in the cat.

SUMMARY.

Employing the silver impregnation method of Del Rio Hortega, the Mallory and the Iron-hematoxylin methods, the author studies the neuroglia of the nuclei of the cochlear pathway (ventral and dorsal cochlear nuclei; superior olive; trapezoid body nucleum and lateral lemniscus nucleum); the sub-cortical acoustic centers (tuberculum quadrigeminus inferior and medial geniculate body) and the cerebral cortex concerning the gyri in front of and behind the pseudo-sylvian sulcus; presumed area of representation of the acoustic radiation; in four cats, intoxicated with daily doses of 0.10 to 0.20 grams of dihydro-streptomycin, applied parenterally for 17 to 22 days. One cat was used as control.

The gliar architecture was found disorganized in the nuclei of the cochlear pathway and in the sub-cortical centers. The neuroglia cells appear with protoplasmatic processes not easily identifiable. In some cases it is visible, the phenomenon so-

called phanerosys and gliar-reaction expressed by intense mitotic activity, which is seen mainly in the geniculate body. Gliar phanerosys and mitosis are also visible in the girus behind the pseudo-sylvian sulcus, permitting the author to assume the hypothesis of this being the cortical center of the acoustic radation.

REFERENCES.

1. Fowler, E. P., and Seligman, E.: Otic Complications of Streptomycin Therapy. *J.A.M.A.*, 134:679, 1947.

2. Carr, David T.; Brown, H. A.; Hodgson, C. H., and Heilman, F. R.: Neurotoxic Reactions to Dihydro-Streptomycin. *J.A.M.A.*, 143:1223, 1950.

3. Shane, S. J., and Laurie, J. H.: Neurotoxity of Dihydro-Streptomycin. *Can. M.A.J.*, 63:277, 1950.

4. Glorig, Aram: The Effect of Dihydro-Streptomycin Hydrochloride and Sulfate on the Auditory Mechanism. *Ann. Otol., Rhinol. and Laryngol.*, 60:327, 1951.

5. Heck, Walter E.; Lynch, William J., and Graves, Harold L.: A Controlled Comparison of the Eighth Nerve Toxicity of Streptomycin and Dihydro-Streptomycin. *Ann. Otol., Rhinol. and Laryngol.*, 62:101, 1953.

6. Caussé: Experiences sur l'Action Toxique Vestibulaire de la Streptomicine. *Comm. a L'Acad. Nationale de Medicine, Bull. Acad. Med.*, Paris, 133:446, 1949.

7. Castaldi, L.: Studi sulla Struttura e sullo Sviluppo del Mesencefalo. *Arch. Ital. Anat. e Embr.*, 23:481, 1926.

8. Beccari, N: Neurologia Comparata. *Sansoni Edizioni Scientifiche*, Firenze, 1943.

9. Pfeifer, R. A.: Der Zentrale Abschnitt der Horleitung. *Abhandl. d. Math. Phys. Klasse d. kon sachs. Gesellsch. d. Wissensch.*, 37:1.

10. Campbell: Referred to by Beccari.[8]

11. Penfield, W., and Jasper, Herbert: "Epilepsy and the Functional Anatomy of the Human Brain," p. 53, 169, Little, Brown & Co., Boston.

12. Walker: Referred to by Beccari.[8]

13. Cajal, R.: "Histologie du Systeme nerveux de l'homme et des Vertebres," 2:634, A. Maloine Editeur, Paris, 1911.

14. Dusser deBarenne: Referred to by Beccari.[8]

15. Brodman: Referred to by Beccari.[8]

16. Rio Hortega: "La Neuroglia Normal. Conceptos de Neuroglioma y Angioglioma." 1942.

17. DeCastro: Modulacion de un Arco Reflejo en el Simpatico. *Trab. del Lab. de Invest. Biol. dela Univ. de Madrid*, 34, 1942.

18. Golgi, C.: Opera Omnia. *Edizione Hoepli*, Milano, 1903.

19. Cerletti, U.: Sulla Struttura della Nevroglia. *Rend. Accad. Lincei*, 5:2, 1915.

20. Loreti F.: Configurazione dell'Oligodendroglia Interfasciculare e

sua Analogia con le Cellule di Schwann. *Riv. Pat. Nerv. Ment.*, 52:135, 1938.

21. BAIRATI, A.: Morfologia e Struttura dei Gliociti. *Biologica Latina*, 2:600, 1950.

22. CONTU, P.: Ricerche sulla Glioarchitettonica dei Vertebrati: Mammiferi. *Arch. Ital. Anat. e Embr.*, 59:2, 1953.

23. PENFIELD, W.: Oligodendroglia and its Relation to Classical Neuroglia. *Brain*, 47:430, 1924.

24. ROBERTSON: A Microscopical Demonstration of the Normal and Pathological Histology of Mesoglia Cell. *Jour. Ment. Sci.*, Oct., 1900.

25. BAUER, K. F.: Das Neurofibrillenbield der Menchlichen Goshinrind. *Zeit. fur. Anatomie*, 112, 1942.

26. SANCEZ IBANEZ, and PEREZ, R.: Les Alterations Hystopatologiques dans l'Encefalite, Etc. *Trab. Invest.*, 30, 1935.

27. STIGLIANI, R.: Esiste Divisione Cellulare della Neuroglia? Iperplasia vera o Apparente nelle Gliosi. *Rass. Biol.*, Umana, 1, 1940.

28. LUGARO, E.: Sulle Funzioni della Nevroglia. *Riv. Pat. Nerv. e Ment.*, 12:208, 1907.

29. CARDONA: Referred to by Bairati.[21]

OBSERVATIONS AND CONSIDERATIONS ABOUT THE COCHLEAR INNERVATION OF THE CAT.

PAOLO CONTU,

University of Recife, Brazil.

Incomplete anatomical bases have been the cause of many hearing theories; therefore, problems concerning cochlear innervation are doubtless important.

In our laboratory of Microscopic Anatomy we are considering the cochlear nerve problem, establishing systematic research under the most modern methods and techniques, and considering not only the organ of Corti but also the whole acoustic system.

Careful revision of available literature demonstrates different distribution of peripheric cochlear nerve fibers among man and other mammals.

Lorente de Nó's[1,2] classical division of nerve fibers in radial, external spiral, internal spiral and intraganglionic spiral fibers, was lately substantially modified by identifica-

tion of the internal spiral with the intraganglionic spiral fibers, whose real origins are in the superior olivar peduncle, forming the so-called Rasmussen bundle.[3,4] These fibers are directed to the internal acoustic cells which, therefore, receive double nerve supply, afferent and efferent.

Rasmussen's description was confirmed by Fernandez[5]; Portmann[6]; Contu and Pirodda[7]; and Freire deBarros[8]; while Bocca[9,10]; Palumbi[11] and Del Bó and Livan[12,13], expressed the opinion that the spiral fibers were from sympathetic or vestibular origin, reminding us of the so-called cochleo-vestibular anastomosis, described by Boettcher,[14] Bovero,[15] Oorth,[16] Weston and Mesolella.[17] Considering the external fibers, Held[18] and Kolmer[19] agree on their course and termination in the external acoustic cells.

Very little emphasis has been placed upon the ganglion of Corti and with the exception of Fort,[20] the majority of writers place the ganglionic cells in the Rosenthal canal. Writers limit themselves to the description of the cochlear nerve as a bundle of axons pertaining to the ganglion of Corti's bi-polar cells. As a rule, neuroglia was found on its first segment, 6 to 8 mm. from its apparent origin.[21]

Many years ago, Hyrtl, Stannius, Kolliker and Pierret[22] found cells on the surface and interior of the acoustic nerve, and Erlisky[23] and Fort,[20] and recently Chiarugi,[24] described cellular elements in the vestibular branch of the VIIIth nerve, arranged in groups as in the principal ganglia.

We used the cat as experimental animal, intravitally fixed with Rio Hortega's or De Castro's formulas. The whole temporal bone was removed with the acoustic nerve up to the rhombencephalon. Specimens were submitted to electrolytical decalcification and silver impregnated, by the Cajal De Castro, Bodian and Rio Hortega metohds. Frozen sections were made from those under Cajal De Castro and Bodian impregnation, and colloidan-paraffin-fast inclusion permitted us to obtain a complete seriation from the cochlear to the rhombencephalon.

We reached the following conclusions:

1. Our findings confirm the majority of those previously reached, mainly those of Kolmer, on the nerve supply of the external acoustic cells. The different fibers form the external spiral bundles of the 1st, 2nd, 3rd and 4th orders; progressively decreasing are the continuation of the radial fibers arising from the lamina espiralis, running radially to the gallery of Corti; and considering their particular topographical arrangement, would be better called "spiro-radial fibers".

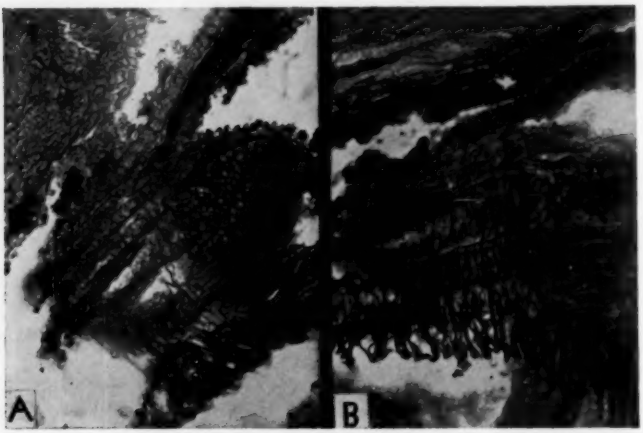


Fig. 1.

Section of the organ of Corti with the external spiral bundles.
(Cajal-De Castro Method—Enl. 500X)

2. The fibers that come from the habenula go to the external acoustic cells through the gallery of Corti by the floor or the top, justifying the previous denomination of basilar and medial. The basilar fibers reach the acoustic external cells, after passing through the Deiters cells, as well as the medial fibers. This is clearly confirmed by Fernandez's topographical distribution of the medial and basilar fibers. Both are found along the whole length of the gallery, but the medial fibers are found in greater number in the basal turn, and are gradually replaced by the basilar fibers that are frankly predominant in the apex.

3. The Rasmussen bundle is very evident on the base of the lamina espiralis, its fibers composing the internal spiral fibers, a continuation of the intraganglionic ones. As previously demonstrated by Fernandez, they do not exclusively innervate the internal acoustic cells, but many turn backwards to the sustaining cells and from there are directed to the base of the limbus espiralis. Portmann[25] already described nervous fibers of sensitive aspect in the limbus laminae espiralis.

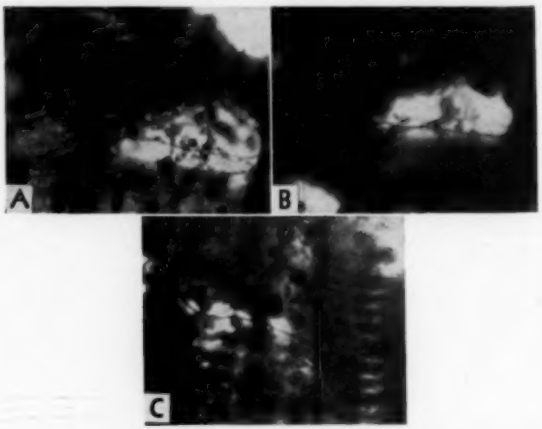


Fig. 2.

(A)—Medial fibers.

(B) and (C)—Basilar fibers passing through the gallery of Corti.
(Cajal-De Castro Method—Enl. 500X)

4. Some of the spiral fibers of the gallery were found going to the apex, and some to the round window, as demonstrated by Fernandez. In our specimens, compact groups of fibers coming out of the foramina were seen, penetrating radially the gallery of Corti, in whose center they were distributed brushlike. Some of those fibers are directed to the external acoustic cells, whose nervous formations could not be followed. Some branches were found reaching the sustaining cells and the sulcus laminae espiralis. Held[18] attributes the innervation of the internal acoustic cells to these.

5. With the Rio Hortega, in some celloidin sections we

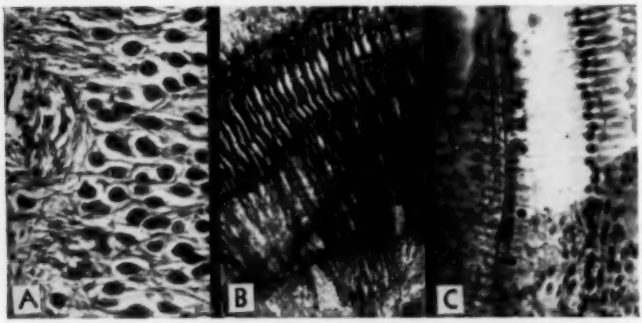


Fig. 3.

(A)—Spiral ganglion. On the left, transversally sectioned, fibers of the Rasmussen spiral-bundle. (Bodian Method—Enl. 500X)

(B)—Bony "Lamina Espiralis" with spiral fibers. (Cajal-De Castro Method—Enl. 500X)

(C)—Base of internal acoustic cells showing double nerve supply. (Bodian Method—Enl. 500X)

Fig. 4.

Spiral fibers of the gallery. (Cajal-De Castro Method—Enl. 500X)

found pyriform nerve cells of the pseudo-uni-polar type, on the vestibular wall, with one axon directed laterally to the base of attachment of the Reisner ligament and of the tectorial membrane; the other, to the sulcus and base of the limbus espiralis. One isolated element of bi-polar type was also found.

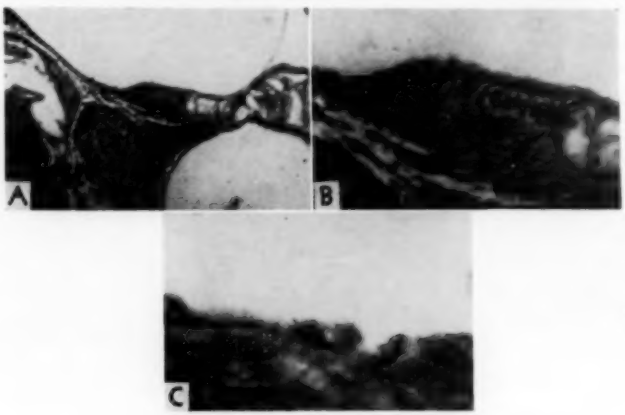


Fig. 5.

(A)—Vestibular wall of the "Limbus Espiralis" with pseudo-uni-polar cells. (Del Rio Hortega Method—Enl. 150X)
(B)—Idem.
(C)—Vestibular wall of the "Limbus Espiralis" with bi-polar cells. (Del Rio Hortega Method—Enl. 500X)

6. Partially confirming and completing Fort's previous description, we assumed in 1956[26] that the cells of the ganglion of Corti are not strictly localized in the Rosenthal's spiral canal. There is a gradual diminishing of the cellular elements starting from the spiral canal to the axis of the modiolus. The ganglionic cells do not remain in the Rosenthal canal, where they are grouped mainly in the medial and internal portion, but as a matter of fact, are seen along the descending canals of the modiolus, essentially in the proximal portion of each canal, and they should not be considered only similar to the cells at the ganglion of Corti but also as migrated cells, and therfore, as a part of it.

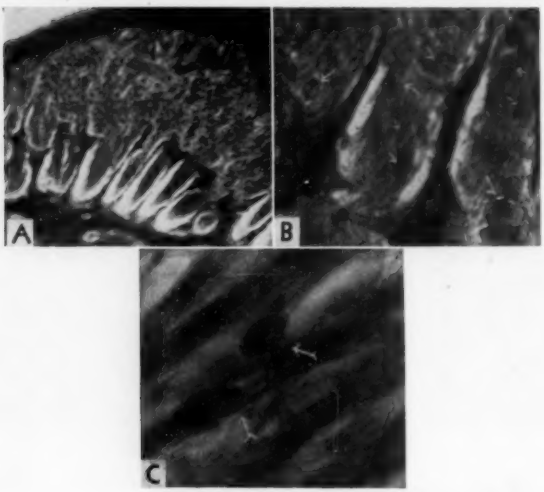


Fig. 6.
(A)—Rosenthal and descending modiolar canals.
(Cajal-De Castro Method—Enl. 85X)
(B)—Bi-polar-cells in a descending modiolar canal.
(Cajal-De Castro Method—Enl. 375X)
(C)—Bi-polar cells in a descending modiolar canal.
(Cajal-De Castro Method—Enl. 850X)

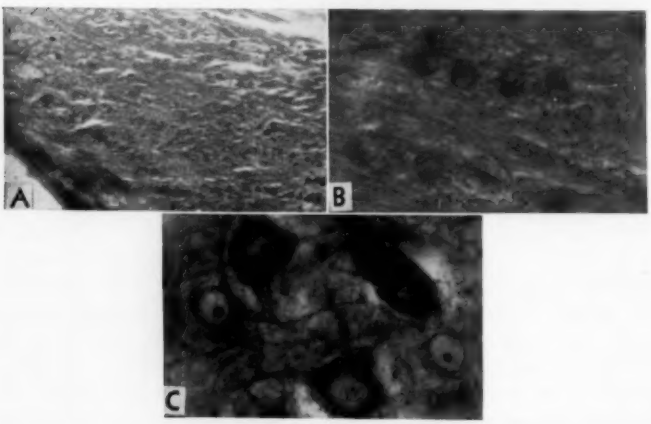


Fig. 7.
(A)—Acoustic nerve with multipolar cells (Enl. 85X).
(B)—Idem.—(Enl. 375X).
(C)—Iden.—(Enl. 850X). (Cajal-De Castro Method)

7. In 1956[27,28] we reported the existence of multi-polar nerve cells in the cochlear branch of the acoustic nerve. Probably those cells were an external projection of the primary acoustic nuclei.

8. By the demonstration made, the complication of the cochlear innervation is evident. The presence of pseudo unipolar type cells in the vestibular wall of the limbus espiralis, suggests the hypothesis of innervation of the tectorial membrane and, therefore, the possibility of closing a "neuronic arch" between that membrane and the hairs of the internal and external acoustic cells.

SUMMARY.

Studying the cochlear innervation of the cat, the author, employing the silver impregnation methods of Cajal De Castro and Rio Hortega, after electrolytical de-calcification, reached the following conclusions:

a. He agrees with Kolmer in the innervation of the external acoustic cells by external spiral bundles of first, second, third, and fourth orders, that by particular topographical disposition would be better called spiro-radial bundles.

b. The Rasmussen bundle is very evident, and some of its fibers turn backwards to the sustaining cells reaching the base of the limbus espiralis.

c. The spiral fibers on the organ of Corti are easily identified.

d. In the vestibular wall of the limbus espiralis were found pseudo-unipolar cells with fibers which were directed to the base of the limbus espiralis and on the other side to the tectorial membrane.

e. The cells of the ganglion of Corti did not remain circumscribed in the Rosenthal canal, but they were also found in the descending canals of the modiolus.

f. In the cochlear branch of the acoustic nerve were found multi-polar cells probably originating in the primary acoustic nuclei.

BIBLIOGRAPHY.

1. Lorente de Nó, R.: Anatomy of the VIIIth Nerve. The Laryngoscope, 43:1, 1933.

2. Lorente de Nó, R.: Symposium: Neural Mechanism of Hearing: Sensory Ending in the Cochlea. *Anat. Physiol.*, 1937.

3. Rasmussen, G. L.: The Olivary Peduncle and Other Fiber Projections of the Superior Olivary Complex. *Comp. Neurol.*, 84:141, 1946.

4. Rasmussen, G. L.: Further Observation on the Termination of the So-called Olivary Peduncle. *Abstr. Anat. Rec.*, 106:235, 1950.

5. Fernandez, C.: The Innervation of the Cochlea. The Laryngoscope, 61:1152, 1951.

6. Portmann, G. eC.: La Double Innervation de l'Organe de Corti. *Acta Oto-Laryngol.*, 43:226, 1953.

7. Contu, P., and Pirodda, E.: Note di Morfologia Sulle Terminazioni Nervose della Coclea di gatto. *Monit. Zool. Ital.*, 61:1-12, 1953.

8. Freire de Barros, H.: Avaliacao Comparativa de Vários-métodos de Impregnacao de Prata, para o Estudo das Células do Ganglio de Corti e do Feixe Intraganglionar. *Tese para Livre Docencia da F.M.U.R., Recife*, 1955.

9. Bocca, E.: Notes on the Innervation of The Cochlea 1°. *A.M.A. Arch. Otolaryngol.*, 55:188-205, 1952.

10. Bocca, E.: Notes on the Innervation of the Cochlea II°. *A.M.A. Arch. Otolaryngol.*, 58:690-703, 1953.

11. Palumbi, G.: Nuovi Apparati Espansionali Nervosi Recettori nel Labirinto Menbranoso dell'Orecchio Umano, 1950.

12. Del Bó e Livan, M.: Nuovi Dati sulla Distribuizione della Fibre Cocleari. *Boll. Soc. Med. Chirur. di Pavia.*, 5:1103, 1950.

13. Del Bó e Livan, M.: De l'Existence d'un Plexe Nerveux Marginal au Niveau de la Lame Spirale. *Acta Anatom.*, 13:16, 1951.

14. Boettcher, A.: Über Entwichelung und bau des Gehorlabyrinths. *Verhlandl. K. Leop. Carol. Deutch. Akad. Naturforscher*, 1869.

15. Bovero, A.: Connessioni Simpatiche del Ganglio Vestibulare del Nervo Acustico. *Arch. Ital. Otologia*, 25:41, 1914.

16. Oort, H.: Uber die Verastekung des N. Octavus bei Säugetierem. *Anato. Anz.*, 51:272, 1928.

17. Mesolella, V.: Sul Cosidetto Ramo Vestibolare del Cocleare e sul suo Ganglio (Ganglio di Boettcher). *Clinica Otolarin., Napoli*, 15:41, 1939.

18. Held, H.: "Handbuch der Hals, Nasen und Ohrenheilkund; Kenker, A., and Kahler, O." Julius Springer, Berlin, 1926.

19. Kolmer, W.: "Handbuch der Mikroskopischen Anatomie des Menchen." Julius Springer, Berlin, Vol. II, I, p. 3066, 1928.

20. Fort, A.: "Traité d'Histologie," 1st Ed., 702. Adrien Delahaye, Paris, 1873.

21. Tarlov, I. M.: Structure of the Nerve Root; Nature of the Junction Between the Central and the Peripheral Nervous System. *Arch. Neurol. Psychiat.*, 37:555, 1937.

22. Testut, L.: "Traité d'Anatomie Humaine," 5th Ed., 98-99, Octave Doin, Paris, 1905.

23. ERLISKY: De la Structure du Tronc du Nerf Auditif. *Arch. de Neurologie*, 1882.

24. CHIARUGI, G.: "Istituzioni di Anatomia dell'Uomo," Vol. IV, Sec. 2: 700-703, Società Editrice Libraria, Milano, 1940.

25. PORTMANN, M. EC.: Innervation de la Bandelette Sillonnée (Limbus Laminae Spiralis) chez le chat. *Experientia*. 8:1, 11-15, 1952.

26. CONTU, P.: Estudo Sôbre Inervacão da Cóchlea e Sobre Novos Dispositivos Nervosos Na-crista da Parede Vestibular do Limbo espiral (no gato). *Anais da Fac. de Med. Univ. do Recife*, 16:1, 1956.

27. CONTU, P.; NEVES PINTO, R. M., E SANTOS, M.: Consideracoes sôbre a Anatomia do ramo coclear do nervo acústico (no felis domestica). *Anais da Fac. Med. Univ. Recife*, 16:2, 1956.

28. CONTU, P.; NEVES PINTO, R. M.: Nota sobre a Tropografia do Ganglio Espiral de Corti (no felis domestica). *Anais da Fac. Med. Univ. Recife*, 16:2, 1956.

DC POTENTIALS OF THE MEMBRANOUS LABYRINTH. (Abstract).*

CATHERINE A. SMITH,
HALLOWELL DAVIS,
BRUCE H. DEATHERAGE,
and
CARL F. GESSERT,

Washington University, and Central Institute for the Deaf,
St. Louis, Mo.

The membranous labyrinth of the internal ear is a closed epithelial tube filled with endolymph and surrounded by perilymph. It is divided into compartments by local constrictions and dilations. The cochlear duct and the saccule are connected by a short narrow duct. The latter is also separated from the utricle by another (indirect) tubule. Previous analysis of endolymph has shown it to contain 144 m. eq./1 of potassium and 16 m. eq./1 of sodium, whereas perilymph shows the opposite potassium-sodium ratio. (Smith, Lowry and Wu).

The DC potentials of the cochlear duct, utricle and saccule of the guinea pig were measured by means of a glass pipette-

*The full paper will be published elsewhere.

electrode. Endolymph was withdrawn in the same pipette for potassium analysis. Successive measurements made on the same ear showed the potential of the cochlear duct to be +70 to +80 mv. (as described by von Békésy), and that of the utricle, to be not more than +5 mv. The saccular potential, measured in other animals, was similar to that of the utricle. Endolymph from both utricle and cochlea had a high potassium content similar to that of the previous analysis. The high positive potential is, therefore, restricted to the cochlea, whereas the high potassium content appears to be the same throughout the membranous labyrinth.

MODIFICATION OF COCHLEAR POTENTIALS PRODUCED BY STREPTOMYCIN POISONING AND BY EXTENSIVE VENOUS OBSTRUCTION.*†‡**

H. DAVIS,
B. H. DEATHERAGE,
B. ROSENBLUT,
Central Institute for the Deaf,
St. Louis, Mo.
C. FERNANDEZ,
R. KIMURA,
University of Chicago,
Chicago, Ill.,
and
C. A. SMITH,
Washington University,
St. Louis, Mo.

INTRODUCTION.

The endolymph in scala media of the guinea pig cochlea shows a positive DC potential of about 80 mv. relative to the

*Prepared under Contract N6onr 272 between Central Institute for the Deaf and the Office of Naval Research.
†Supported in part by research grant No. B-966 (C) from the National Institute of Neurological Diseases and Blindness, Public Health Service.
‡Reproduction in whole or in part is permitted for any purpose of the United States Government.
**Assisted by U. S. Public Health Service Grant B-680.

perilymph and surrounding tissues. This DC endocochlear potential is thought[1,2] to contribute to the generation of the cochlear microphonic and to the associated electrical current flow that is believed to stimulate the nerve endings.[4] The source of the DC potential is unknown. The electrochemical mechanism seems to be rather specific and more closely dependent on oxidative processes than is the familiar negative intracellular polarization.[4] The site of the generation had been tentatively located both in the hair cells and in the stria vascularis. The present experiments were planned in the hope of deciding between hair cells and stria vascularis as the locus, by injuring the inner ear of guinea pigs in ways which could be more or less selective. The experiments actually yielded important additional information concerning the source and significance of both the cochlear microphonic and the negative summating potential.

As an agent to injure hair cells, streptomycin, injected locally into the bulla, was suggested to us by Dr. Harold Schuknecht.[11] He actually made the first three injections in the present series. The procedure directed at the stria vascularis was extensive obstruction of the cochlear vein and some of its collaterals. Kimura and Perlman have described the surgical procedure and the resulting pattern of histological changes in stria vascularis and in the sensory cells.[7] The earliest changes apparently occur in the stria and the spiral ligament.

We did obtain clear differential effects by these two methods. In each case an interval of a few (four to seven) days proved best for obtaining the desired degree of change in electrical activity and in histological appearance.

The electrical responses measured, as indices of the functional state of the organ of Corti, were the cochlear microphonic (CM), the negative summating potential (SP), and the action potential spike (AP). CM is known to depend upon the hair cells. AP is generated by the nerve fibers in the modiolus and gives evidence of the integrity of the overall system of tectorial membrane, hair cell, nerve ending and nerve fiber. SP, like CM, probably originates in hair cells, but the association has not been established clearly. To draw

firm conclusions, therefore, we seek primarily a relation between CM response and the positive DC resting potential of scala media under varying degrees of injury inflicted by the two different methods. Secondly, we seek the relation between depression of electrical activity and any histological changes that can be demonstrated in particular types of cell. Finally, the relationships among the various electric responses are of great interest in their own right, as they indicate possible mechanisms or functional relationships.

METHODS.

Streptomycin sulfate, in doses of 0.1 to 1.0 gm. dissolved in distilled water, was injected into one bulla of each of 26 guinea pigs. The volume of fluid injected was usually about 0.2 cc. The hypodermic needle was at first forced through the wall of the bulla or through the tympanic membrane. It proved more satisfactory, however, to expose the bone of the bulla in a preliminary operation and to drill one hole for the hypodermic needle and another to allow escape of air. Even so, some fluid may have been lost through the Eustachian tube before the drug was entirely absorbed, so that the exact dosages were uncertain.

A time interval of four to seven days between injection and test was convenient and sufficient. Longer intervals increased the chances of complications, such as otitis media and formation of adhesions, and tended to give more injury to the cochlea than was desired.

Practically all 22 of the animals successfully injected and carried to acute experiment, showed signs of unilateral vestibular deficit, such as nystagmus and a head posture with rotation around the long axis, during the first two days of the interval.

At the final experiment the bulla usually contained some mucoid or serous blood-stained fluid, and the mucosa was often thickened and vascularized; but the tympanic membrane was intact and, after the bulla had been swabbed out, the ossicles were usually freely movable.

Surgical obstruction of the cochlear vein and some of its

collaterals was carried out by R. K. at the University of Chicago. The method, the resulting symptoms and the usual histological changes have been described elsewhere.[7] The animals were transported to St. Louis and tested at intervals ranging from five days to 28 days. The most satisfactory degrees of partial injury were found five to eight days post-operatively.

Both the streptomycin and the venous-congestion animals were prepared for final test by our usual methods.[13] Dial in urethane 0.5 cc. per kilo was given intraperitoneally. Rarely, a small supplementary dose was required. The treated cochlea was exposed by opening the bulla and two fine, enamel-insulated nichrome-steel wires (25 μ) were introduced through small holes drilled into scala vestibuli and scala tympani respectively 3 to 4 mm. from the round window. Between these electrodes a fenestra was made through the bony shell of the cochlea to expose the spiral ligament behind the stria vascularis. A chlorided silver electrode, wrapped in ringer-soaked cotton to avoid direct contact with the body fluids, was inserted under the skin at the edge of the wound and, in most experiments, an ear speculum that fitted snugly the output tube of a calibrated sound source[3] was sewed tightly into the external auditory canal.

The first four streptomycin animals were simply placed at a standard distance in front of an open loud-speaker and tested qualitatively with 9000 c.p.s. tone bursts, but it was soon found that stronger acoustic stimuli were needed. The closed, calibrated system, capable of delivering over 135 db. SPL at the test frequency of 7000 c.p.s., was employed thereafter, and all sound pressure levels readings are corrected to a position just in front of the tympanic membrane.

As a standard acoustic stimulus we employed a 7000 c.p.s. tone-burst with a 1 msec rise time and 1 msec fall time. The sound level remained constant for 4 (or sometimes 6) msec. This interval was long enough to allow easy recognition of SP but too short for echoes in the acoustic system to confuse the pattern. The 1.0 msec rise time is the slowest that we found consistent with the good synchronization of nerve impulses that is necessary for a sharp "N_1 spike."

Complete input-output functions for cochlear microphonics, action potential and negative summating potential were plotted from measurements made of standing wave patterns on the face of the oscilloscope. The results are expressed as microvolts DC displacement from the resting baseline for the summating potential, as microvolts peak-to-peak for the cochlear microphonic and as microvolts relative to the baseline of the first wave (N_1) in the action potential response. CM and SP were both measured as voltage difference between scala vestibuli and scala tympani. AP was recorded between the null point (for CM) on a resistance connected between the scala vestibuli and the scala tympani electrodes and the neck electrode. Adjustment of the variable resistances in the network effectively eliminated CM potentials from the AP trace and vice versa.[13]

With these electrode positions the AP is recorded as the impulses enter the internal auditory meatus, and the spike represents the impulses in *all* fibers that are activated within the first millisecond, regardless of the point of origin in the cochlea. The CM and SP responses and the resting DC polarization, however, are chiefly those generated within one or two mm. of the position of the electrodes. The contribution from distant points falls off rapidly with the distance.[13,14] The attenuation of CM with distance may be even more rapid than for the DC potentials because of capacitative conduction across the walls of the scala media. The locus of maximal amplitude of displacement of the cochlear partition at 7000 c.p.s. is only slightly apical from the position of the recording electrodes[12,13] so both CM and SP are recorded efficiently.

After recording the input-output data, we measured the DC potential of the interior of scala media relative to the outer surface of the spiral ligament. The head of the animal had been clamped firmly in a standardized position. A micropipette electrode was now advanced into the scala media by means of a micro-manipulator. Visual control was maintained through a binocular dissecting microscope (20x). Pipettes about 15 μ outside diameter filled with 0.85 per cent Na Cl solution gave a good compromise among the factors of simplicity of preparation, low enough ohmic resistance, me-

chanical stiffness, and ability to penetrate spiral ligament and structures beneath it without undue injury. Details of the construction of the pipette-electrode, its carrier and the associated DC amplifier are given elsewhere.[12] Measurements were made on the face of an oscilloscope with the help of an internally illuminated lucite scale. A moving-film record of the movements of the oscilloscope spot during insertion and withdrawal of the pipette was made as routine but was actually used only to resolve doubts as to direct scale readings when the changes were unusually complicated or rapid. The silver electrodes were freshly chlorided each day.

Just before penetration the amplifier was always balanced to give zero reading while the tip of the pipette was immersed in the film of fluid over the spiral ligament. A zero reading was also taken again after withdrawal but just before breaking contact. This final reading usually agreed within $\pm$ 1 mv. with the original. The pipette was advanced by steps until further advance gave no further increase in DC potential. In a few cases the last advance caused a fall in the measured potential, presumably due to penetration of Reissner's membrane. Only rarely did we fail to encounter a region of strong positive DC potential on the first attempt. Always when the maximum was less than +30 mv. and sometimes, as a control, in other experiments a second and even a third penetration was made in a slightly different position or direction. Actually, if a potential over 30 mv. had been encountered on the first trial no higher potential was found on any later trial, and usually the voltage was the same or a few millivolts less.

As control values for DC measurements made in the basal turn by the above method we employed nine initial measurements made in another series of experiments that involved subsequent chemical injections into scala media or scala tympani.[5] These experiments overlapped the present series in time.

Normal thresholds and input-output curves were determined in a series of 16 experiments that will be described in detail elsewhere. The procedures were identical with those of the

present series except that no fenestra was made in the basal turn, and sometimes a second pair of recording electrodes was placed in the third turn.

At the end of the acoustical and electrical tests the animals were perfused with isotonic sodium chloride solution introduced into the aorta. After exsanguination the animal was perfused with Heidenhain-Susa solution and the head or the temporal bones prepared for histological study by routine methods. The heads of the animals with venous obstruction, studied by C. F. at the University of Chicago, were sectioned as a whole through both cochleas symmetrically.[7] This method of cutting requires careful orientation in the microtome to obtain mid-modiolar sections of both cochleas at once, but allows close immediate comparison between the operated and the opposite normal ear.

RESULTS: HISTOLOGICAL.

Streptomycin.

The injurious effects of streptomycin varied according to the dosage employed, the number of days elapsed following the dosage, the type of tissue, and the distance from the round window. The relationships of injury to size of dose and to duration following the injection were not examined closely because it was obvious that only slight correlations exist within the range of dosage represented in our experiments. The external hair cells are the most vulnerable cells. The supporting structures of the organ of Corti and the internal hair cells are fairly vulnerable, the stria vascularis is affected only slightly, and Reissner's membrane, the basilar membrane, sulcus cells and limbus are very slightly affected, if at all. A gradation of effect away from the round window is very clear and seems to establish beyond doubt the route of entrance of the streptomycin, namely through the round window membrane.

The ears studied in this series were examined both with relation to the extent lengthwise of the injury and to the severity of injury in the region of the fenestra. As noted above, our electrode system is sensitive to CM, SP and DC

generated within one or at the most a very few millimeters. The condition of structures three to four mm. from the round window is critical, while the condition of cells in the apical turns is irrelevant for the interpretation of our electrical tests except for the action potentials produced by "remote response."[6] The histological findings and also the results of our electrical measurements are summarized in Table I.

In the streptomycin series, the mildest degree of injury to the hair cells was a slight pyknosis and shrinkage of the external hair cells in the lower half of the basal turn. All but one of these ears, however, showed complete loss of the external hair cells up to, or nearly up to, the position of the recording electrodes. The inner hair cells near the electrodes were sometimes present and apparently normal in appearance (see Fig. 1). In other cases the nuclei were pyknotic and frequently displaced, and, in the most severely injured ears, the internal hair cells as well as the external hair cells (see Fig. 2), and sometimes the supporting structures also, disappeared entirely.

The changes in the stria vascularis were much less than those in the organ of Corti. The most severe change was a shrinkage of cells with some edema appearing between or behind them (see Fig. 3). There was no general cellular degeneration.

Changes in the stria and also changes in the hair cells were not necessarily continuously graded but might vary irregularly from section to section. Whereas all of the ears in the series showed definite changes in the external hair cells, only six showed recognizable changes in stria vascularis, and only one of these showed injury in the stria that extended throughout the length of the cochlea.

In the seven most severely poisoned ears the supporting cells of the organ of Corti had also degenerated. Sometimes they still formed a continuous layer (1072, 1078, 1090, 1108) but in others there were bare patches on the basilar membrane (1073, 1089). In the most advanced cases (1071, 1089) replacement of the degenerated supporting cells by squamous epithelium had begun but was not yet complete. In the

GUINEA PIG NUMBER	TREATMENT	HISTOLOGICAL IN BASAL TURN: STRIA VASCULARIS	HISTOLOGICAL IN BASAL TURN: NEAR ELECTRODES HAIR CELLS INTERNAL	HISTOLOGICAL IN BASAL TURN: NEAR ELECTRODES HAIR CELLS EXTERNAL	RESTING DC MV	ELECTRICAL RESPONSES TO 7000 CPS BURSTS CM DB - SPL Thresh.	CM μV Break	CM μV Max.	SP DB re .0002 dyne	SP μV at 10db	AP DB re .0002 dyne Thresh. 37db	AP μV at 110db	GUINEA PIG NUMBER
	NORMAL MEDIANS				N=9 77 N=8	35	76	103	800	63	500	26	100 400
	STREPTOMYCIN							FIELD STIMULATION					
1071	12 .12	1 Slight local changes	ABSENT in all turns	ABSENT	5 to 10								1071
1078	12 .06	NORMAL	Mostly ABSENT	ABSENT	90 to 105			No responses to 8000 cps bursts					1078
1073	7 .16	1 Moderate local changes	Entire ORGAN of CORTI	ABSENT in all turns	< 4			at	above				1073
1076	7 .07	2 Moderate injury	ABSENT	ABSENT	95				100 db				1076
1080	8 .20	1 Very slight changes	ABSENT in all turns	ABSENT	5								1080
								CLOSED ACOUSTIC SYSTEM					
1079	7 .07	= NORMAL	Minimal injury ‡	Hyaloid and shrunk	87			105		present		good response*	1079
1090	8 .06	NORMAL	FAIR	ABSENT : transition zone	77			very small		present		present	1090
1094	4 .06	NORMAL	ABSENT	in all turns ABSENT	60	113		?	2	but seen at 110 db			1094
1097	4 .10	NORMAL	FAIR	ABSENT : transition†	81	78	99	107	36	78 256	64	0 152	1097
1099	4 .04 Conductive 7.0b	Specimen damaged in preparation			90	49	86	104	264	62 280	38	34 256	1099
1101	3 .04	NORMAL	FAIR	ABSENT below, 6000 above	79	38	70	95	280	51 320	32	36 138	1101
1107	4 .05	2 Marginal but mild	Shrunk	ABSENT : transition	58	64	86	102	60	75 208	80	0 190	1107
1109	4 .05	NORMAL	ABSENT (pyknotic in Turn 2)	ABSENT	73	120	-	-	?	absent at 110db	65 remote AP 54		1109
1110	4 .07	NORMAL	NORMAL	ABSENT, but few in Turn2	72	70	85	105	44	74 160	70	0 60	1110
1111	4 .08	NORMAL	NORMAL	Transition	67	50	?	105	120	59 200	38	54 250	1111
1112	4 .08	NORMAL	Shrunk	Pyknotic †	62	43	72	97	210	58 440	38	56 190	1112
1113	4 .08	NORMAL	NORMAL*	Transition	73	40	68	85	144	50 440	30	114 260	1113
1114	5 .07	NORMAL	NORMAL	Transition	93	51	74	110	174	58 432	45	36 266	1114
1123	6 .10 Otitis Media	NORMAL	FAIR	ABSENT: transition above	88	44	75	95	286	48 500	34	60 240	1123
			6000	ABSENT	86			Very small responses : also conductive loss					
1124	6 .13	NORMAL°	Slightly shrunk	ABSENT; transition nearby	78	85	107	65	78	180	78	C 114	1124
1125	6 .20	1 NORMAL°	ABSENT	in all turns ABSENT	71			No responses of any kind at 130 db					1125
	VENOUS OBSTRUCTION				78 hr MV 77 hr MV								
1103	7	4 Severe DEGENERATION	NORMAL	Severe DEGENERATION	-8.6 36	65	no	107	12	78	64	47 24	240 1103
1104	8 Otitis Media? 15db	3 Moderate DEGEN.	NORMAL	Mild Degeneration	-3.2 53	75	95	91	18	84	100	46 0	180 1104
1115	2lb	3 Partial ATROPHY	NORMAL	NORMAL	-0.8 70	35	70	87	340	55 540	22	144 400	1115
1116	6	4 Transition to degenerate	NORMAL	NORMAL	-5.3 42	36	72	95	340	62 250	53	16 196	1116
1117	2lb	NORMAL	NORMAL	NORMAL	-0.8 70	28	71	98	780	53 420	18	118 540	1117
1118	22 Otitis Media? 15db	NORMAL	NORMAL	NORMAL	-1.1 68	38	74	105	840	60 1000	31	70 690	1118
1119	8	1 Worse above and below	NORMAL	NORMAL	-8.6 36	42	76	101	360	68 208	40	56 380	1119
1120	5	5 ATROPHY + NECROSIS	NORMAL	Very few remain	-14.2 15	13	no	seen	0	no	remote AP	0	1120
1121	5	5 DEGEN. 1mm below	NORMAL	NORMAL	-13.1 17	11	71	107	63	76 134	66	0 112	1121
1126	15	5‡ Spotty DEGENERATION	NORMAL	NORMAL	-1.4 65	32	72	96	880	50 640	20	140 520	1126

TABLE I.

Δ The numbers in this column indicate degrees of abnormality of stria vascularis. No entry = normal. 5 = complete degeneration. Details in text.

* This correction has been applied in deriving the values of electrical data that are printed in small figures.

o Reissner's membrane has collapsed over parts of the stria vascularis but elsewhere it is normal.

† In this and other transition zones the external hair cells are absent below, and present but not entirely normal above the electrodes.

‡ The organ of Corti has collapsed in this preparation, but this is probably an artifact.

severely affected ears some degeneration appeared in the limbus also, and the internal and external spiral sulcus cells were lost.

In one ear (1125) the cochlear duct was collapsed and Reissner's membrane lay closely against the stria vascularis over the greater part, although not quite all, of its width. All

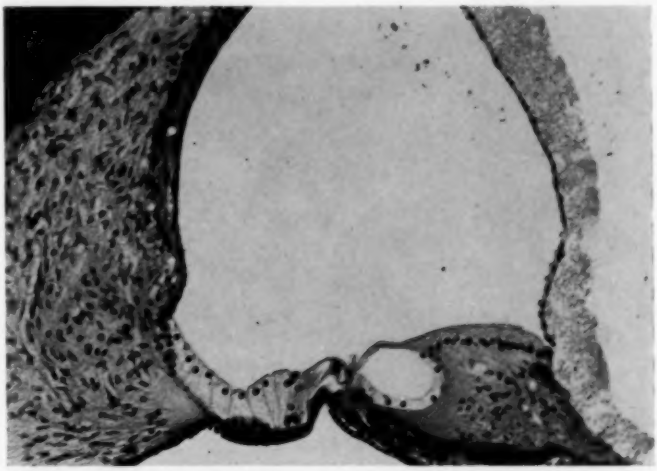


Fig. 1. Degeneration of external hair cells in streptomycin poisoning. Internal hair cell present, in fair condition. Stria vascularis is normal. DC potential +81 mv. See Fig. 10 for input-output curves (Guinea pig 1097).

of the vestibular membranous labyrinth was likewise collapsed in this ear, even the semicircular canals.

In some preparations Reissner's membrane bulged somewhat into stria vascularis, either locally or throughout the cochlea, but we attribute this bulging to artifacts of fixation.

With the exception of animal 1107 no neural injury was observed, either to the nerve fibers or to the ganglion cells, unless the animal had survived at least six days after the injection. After the sixth day the amount and severity of injury to nerve tissues seems to correlate quite directly with

Fig. 2. Degeneration of both external and internal hair cells in streptomycin poisoning. Supporting cells present but abnormal. Stria vascularis is normal. Folding of basilar membrane is a fixation artifact. DC potential +73 mv. See Fig. 12 for input-output curves (Guinea pig 1108).

Fig. 3. Mild injury of stria vascularis in streptomycin poisoning: the most severe in the entire series. DC potential +58 mv. (Guinea pig 1107).

the amount of injury to the hair cells. It seems reasonable to suppose, therefore, that the injury to nerve tissues is not primary but is secondary to degeneration of the hair cells.

Extensive Venous Obstruction.

Satisfactory histological preparations were made of the cochleas with extensive chronic venous obstruction. In general the effects were more severe in the animals studied within eight days following the operation. Recovery apparently occurred after establishment of collateral circulation unless, as in some cases not studied electrically, part of the cochlea degenerated completely.

The histological changes were always most severe in the immediate neighborhood of the round window. The apical turns appeared quite normal and, just as with the streptomycin poisoning, the electrodes in several cases were placed in or very close to a zone of rapid transition from "nearly normal" to "clearly abnormal" tissues. Unfortunately, this gradation of injury makes inferences as to the relation of electrical potentials to particular structures less secure than we had hoped.

The tissue most severely affected by venous obstruction was the stria vascularis. Next in order came external hair cells, the spiral ligament, and the supporting structures of the organ of Corti. Slight hemorrhages into the perilymphatic spaces were frequent. Other tissues were not affected.

The obvious changes in the stria vascularis were: *a.* atrophy of the epithelium from the normal pseudostratified columnar to a simple cuboidal epithelium; *b.* thrombosis or absence of the blood vessels, and *c.* sometimes a detachment of whole cells or extrusion of pigment granules (see Figs. 4, 5).

In Table I the degree of injury to the stria vascularis is coded as follows:

1. Some slight changes in a few cells.

2. Most cells shrunken with some edema between or behind them, but no general cellular degeneration.

3. Atrophy of some cells but not more than 50 per cent.

4. More than 50 per cent of cells atrophic, usually accompanied by necrosis.

5. Almost complete necrosis. (Complete necrosis of stria vascularis was not seen in this series.)

Grades 1 and 2 are characteristic of the streptomycin series. The changes occurred usually near Reissner's membrane or the spiral prominence where a few cells might be degenerated

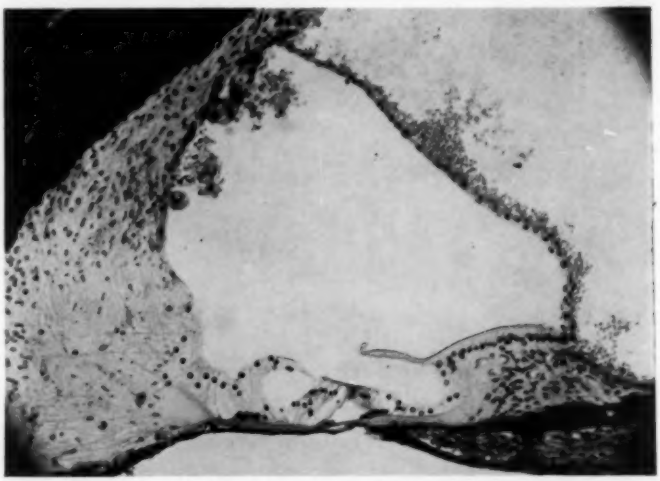


Fig. 4. Degeneration of stria vascularis, Grade 5, with normal hair cells and other structures of the organ of Corti. DC potential +17 mv. See Fig. 9 for input-output curves (Guinea pig 1121).

or sloughed off. These degrees of injury appear compatible with normal function. Grades 4 and 5 were seen only in the venous obstruction animals, in six of which the stria vascularis was injured more severely than in any streptomycin animal. With such injuries normal function would be very improbable or impossible. The atrophy, when present, tended to be localized near the round window while the necrosis was usually quite extensive. In Table I the numbers indicate the degree of injury at the position of the electrodes.

Fig. 5. Atrophy and necrosis of stria vascularis, Grade 5. Internal hair cells present. External hair cells mostly absent. DC potential +15 mv. (Guinea pig 1120).

With venous obstruction the changes in the hair cells were pyknosis or total absence of nuclei. In the spiral ligament nuclei were sometimes missing, and scattered macrophages appeared. All of these changes and their distribution are similar to those already described elsewhere.[7]

In one ear (1116) with normal hair cells and only local degeneration of stria vascularis near the round window, Reissner's membrane was partially collapsed. In another, with normal hair cells and a local region of mild degeneration of the stria, Reissner's membrane was torn from round window to the third turn. The electrical responses of this cochlea were emphatically normal (1117) and the damage is interpreted as an artifact. There were no other suggestions of abnormalities of hydrostatic pressure.

The cochlear aqueduct was not normally entered during the operation producing obstruction of the cochlear vein, but histological examination showed that it had been blocked in five experiments (1103, 1104, 1115, 1118 and 1119). In none of these, however, was there any abnormality of structure or electrical response that could be attributed to the blockage. Animals 1115 and 1118, with survivals of four and three weeks, showed blockage of the aqueduct with new bone formation but they also had normal histology and electric responses. This is clear evidence that the cochlear aqueduct is "dispensable" in the guinea pig.

RESULTS AND INFERENCES: ELECTRICAL.

Endocochlear DC Potential.

The endocochlear DC potential of nine normal controls ranged from +64 to +90 mv. The mean, the median and the mode of this group were all +77 mv. The distribution was normal in form as far as one can tell with such a small sample.

The ears injected with streptomycin divided clearly into two groups. In three animals (1071, 1073 and 1089) the endocochlear potential was 10 mv. or less. From none of these three ears was any electrical response (CM, SP or AP) obtained with 9000 c.p.s. tone bursts at 65 db. above the

normal threshold. In all three the hair cells were absent in all turns, and the entire organ of Corti showed severe degeneration in the first turn. In 1073 and 1089 there were bare patches of basilar membrane, and in 1071 a very thin squamous epithelium had almost, but not entirely, replaced it. The stria vascularis, however, showed only the mildest degree of abnormality in patches here and there.

These three ears do not help to identify the generator of the DC potential. The potential may have been low because the generator in the organ of Corti was destroyed, or else because an intact generator in stria vascularis was short-circuited by a low-resistance pathway through denuded or incompletely covered basilar membrane. The basilar membrane and the mesothelial cells on its tympanic surface alone do not seriously obstruct the movement of ions.[14] The electrical barrier is the organ of Corti, the cells of Claudius and the sulcus cells.[12]

The other 19 streptomycin-treated ears are more decisive. All showed normal or nearly normal endocochlear DC potentials. Only three, at +58, +60 and +62 mv., lay below +64 mv., the lowest of our nine controls. Four showed potentials higher than +90 mv., the highest control. The entire distribution is shown graphically in Fig. 6. Here the abscissa is the "threshold" of the cochlear microphonic response. We shall see below that the voltage of the microphonic correlates very closely with the degree of injury to the external hair cells.

In 1108 a questionable CM response was seen, but only at 129 db. SPL. All hair cells, external and internal, were absent in Turn I, but DC was +73 mv. Stria vascularis was normal. In 1096 a doubtful CM appeared at 113 db. SPL. All hair cells were missing in all turns but the DC was +60 mv. Again stria vascularis was normal. The two ears (1072, 1078) that showed the highest DC potentials, +92 and +95 mv., gave no CM, SP or AP at 100 db. SPL (9000 c.p.s. burst). The hair cells were nearly or completely absent in Turn I in both ears. Stria vascularis was entirely normal in 1072 and nearly so in 1078. On the other hand the ear (1107) with

the lowest DC potential in the streptomycin series (+58 mv.) showed the most definite and widespread injury in the stria vascularis (see Fig. 3). The injury was not severe, however, in comparison with those produced by venous congestion.

The other ears showed normal DC potentials with varying degrees of elevation of their CM "threshold".

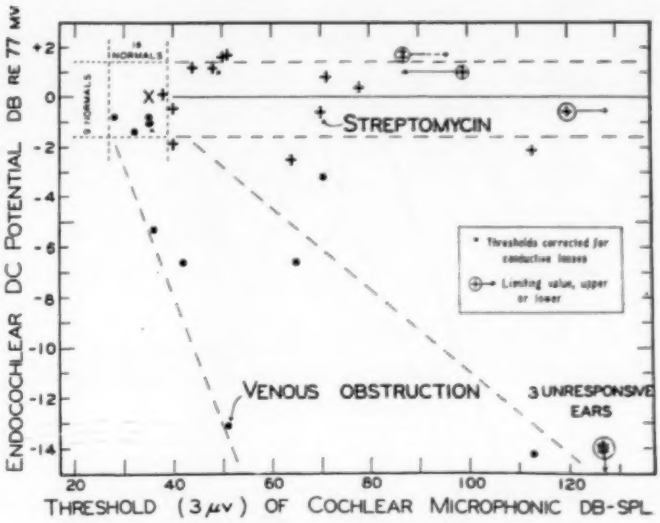


Fig. 6. Distribution of endocochlear potentials with respect to cochlear microphonics. The large X indicates the median values of two groups of normal ears.

Obviously, the DC potential can exist quite independently of the external hair cells and the CM response, but a normal or nearly normal stria vascularis is always present.

The ten ears in which venous obstruction was produced divide into three groups with respect to their DC potentials. Four were within normal limits, although all ten were below the control median. Two (1120 and 1121) were greatly depressed, while the other four showed intermediate, moderately depressed potentials. The four best ears had all been ob-

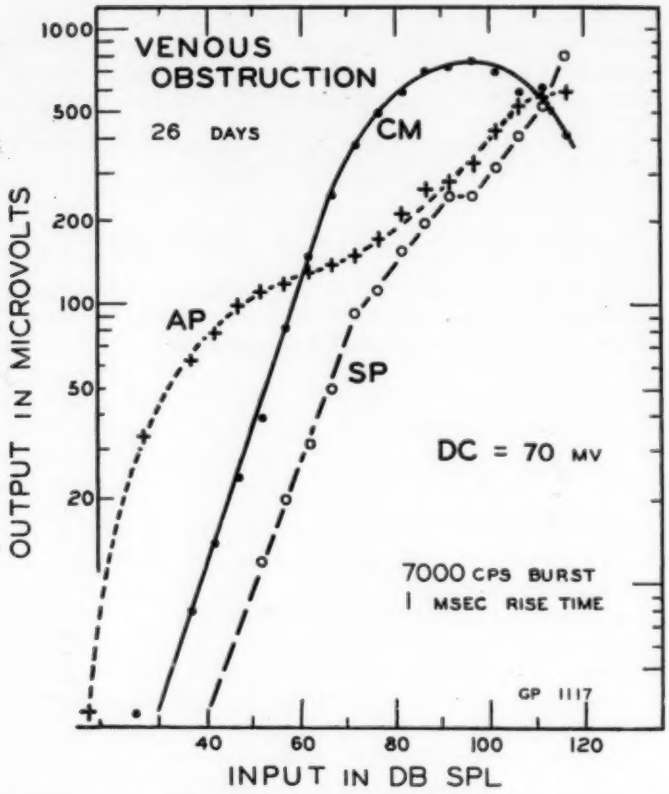


Fig. 7. In this and subsequent graphs of input-output functions the "threshold" response, plotted at 3μv has sometimes been interpolated or extrapolated. In Table I, however, the threshold is the threshold of recognition of the response on the oscilloscope and sometimes differs slightly from the plotted value. Stimulus: 7000 c.p.s. tone burst with 1 msec rise time. Electrodes in the basal turn in all cases. In Guinea pig 1117, with venous obstruction, the input-output relations were substantially normal.

structed at least 15 days previously, and in general their electrical responses were all within normal limits except for a reduced CM maximum voltage in one case. The histological findings were also normal.

In one of the depressed ears (1120) the electrical responses

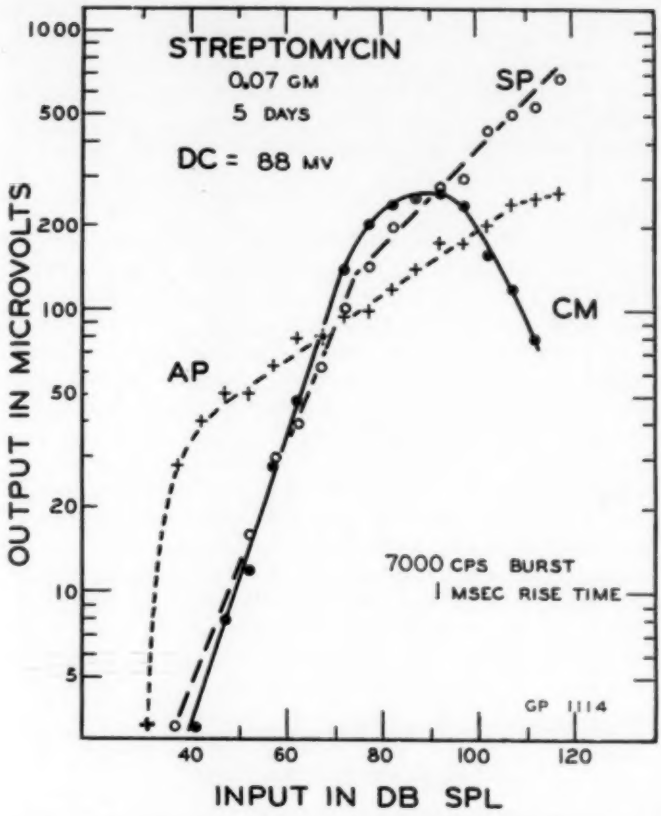


Fig. 8. Normal AP and nearly normal SP input-output curves with slightly depressed CM from an ear poisoned with streptomycin. DC potential is normal.

as well as the endocochlear DC potential were very greatly reduced. The latter was only +15 mv. The internal hair cells were apparently normal. The external hair cells were missing, and the stria was atrophic above and necrotic below the position of the electrodes.

The other depressed ear (1121) showed nearly normal hair cells, but a severe degeneration of stria vascularis from the

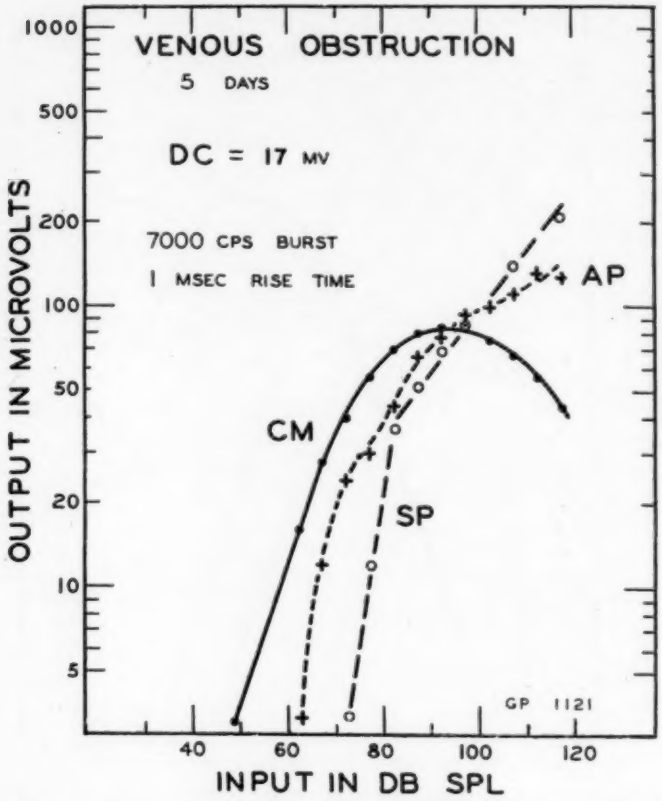


Fig. 9. Moderately depressed input-output curves with very low DC endolymphatic potential in an ear with venous obstruction. The degenerated stria vascularis and intact hair cells are shown in Fig. 4.

round window to within 1 mm. of the electrodes (see Fig. 4). The latter were located in a transition zone between severely and mildly degenerated stria. The electrical responses (see Fig. 9) were definitely but only moderately depressed. The DC potential, however, was only +17 mv.

Ear 1121 with venous obstruction, and ear 1096 in the streptomycin series, form a perfect contrasting pair. They

show that the DC potential may be practically normal with no hair cells present but good stria vascularis, and that even with normal hair cells it may be very low if the stria vascularis is severely injured. *All of our experiments are consistent with the interpretation that the endocochlear DC potential is generated by the stria vascularis and not by the hair cells.*

The Cochlear Microphonic.

The form of the input-output function is very stable. The output is linearly proportional to the instantaneous acoustic pressure at low and moderate levels, but goes through a maximum at a high level. (Fig. 7 illustrates a normal CM response curve.) The sound pressure levels at which the first deviation from linearity appears and at which the maximum is reached depend on the acoustic frequency of the stimulus and on the position of the recording electrodes. For our control series the median values were 76 and 103 db. respectively. These levels are so stable, even when the output voltage is greatly depreseed by streptomycin or by venous obstruction, that we were able to infer from elevated values in three cases (1099, 1104 and 1118) that there were conductive losses of 7, 15 and 15 db. respectively. Otitis media with adhesions had been noted at operation and/or was demonstrated by microscopic examination in each case. (In Table I it is the corrected values that are entered.)

From the stability of the shape of the CM response curve and of the sound pressure level that elicits maximal response, even when the hair cells are injured and the CM output voltage is reduced, we infer that the shape of the upper part of the CM response curve is mechanically determined by structures and forces outside the hair cells.[4]

A third convenient index of CM response is the threshold of visibility of the CM on the oscilloscope. This value agrees very well with the sound pressure level that yields a 3 μv peak-to-peak response as determined by extrapolation from measurements at higher level.

The final index of CM response is the maximum peak-to-peak output voltage. Our median control value is 800 μv.

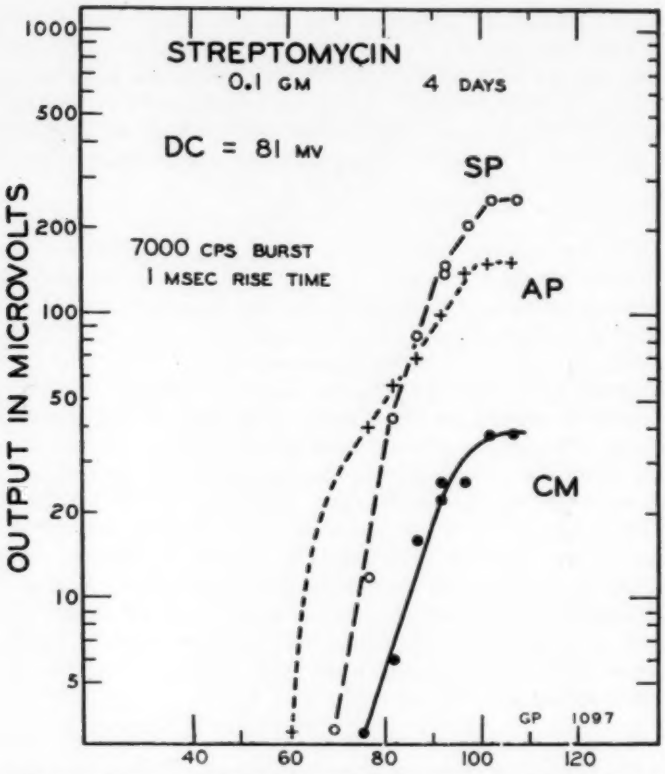


Fig. 10. Depressed responses, particularly CM, in the presence of normal DC endolymphatic potential following treatment with streptomycin. The degenerated external hair cells of this ear are shown in Fig. 1.

The values for all four indices for the individual ears and for the medians of the control series are given in Table I, and full curves are presented in Figs. 7 to 12 for six ears that represent various degrees of depression of cochlear microphonic, summating potential and action potential.

The cochlear microphonic has for many years been attributed to the hair cells.[4] This interpretation is confirmed without exception in the present experiments if we confine the statement to the *external hair cells*. The streptomycin

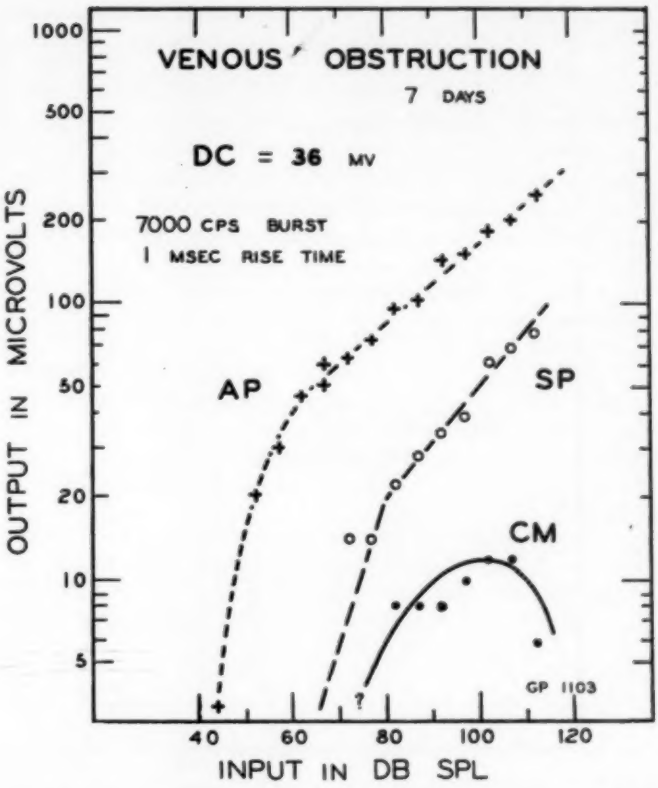


Fig. 11. Input-output curves from an ear with venous obstruction. This ear showed the strongest AP response relative to CM in the entire series. DC potential is depressed.

series offers many examples of this correlation, and the venous obstruction series adds three more.

In every case in which no external hair cells remained (within three millimeters of the electrodes) the maximum CM was not more than 3 μv. In these cases when a trace of CM remained the threshold was elevated by at least 75 db. In other ears, which yielded depressed CM responses five or ten per cent of the normal maximum, and with thresholds

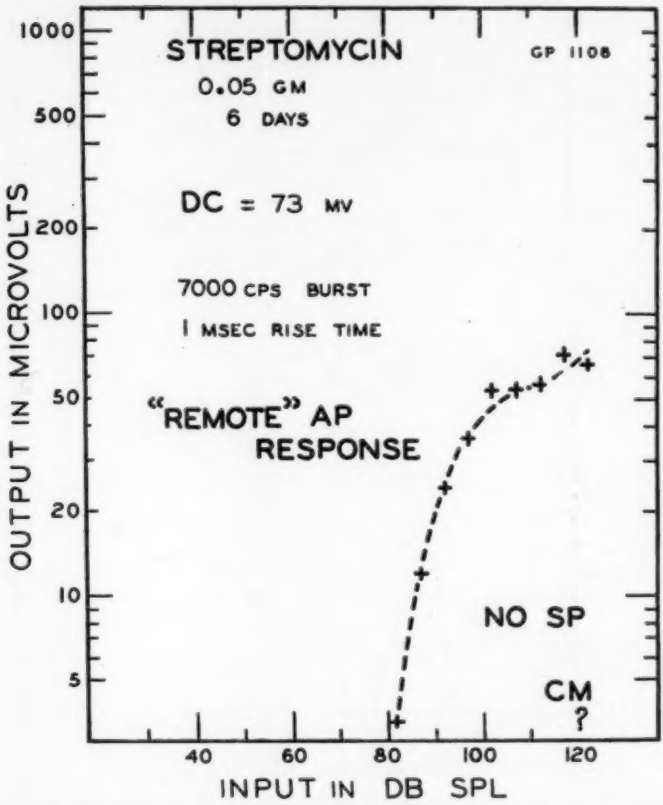


Fig. 12. Following treatment with streptomycin the first turn of this ear showed no local responses. A typical input-output curve was obtained for the "remote" component alone of the AP response. The degenerated hair cells in the basal turn are shown in Fig. 2. Hair cells were still present in the apical turns. The DC potential was normal.

elevated 30 db. or more, either the external hair cells were badly shrunken and pyknotic in the first turn or, more usually, the electrodes had been placed squarely in the transition zone between absent external hair cells toward the round window and some external hair cells, perhaps with pyknotic nuclei, present within one millimeter apically. Apparently a shrunk-

en hair cell with a pyknotic nucleus may generate a significant, though perhaps subnormal, cochlear microphonic.

The internal hair cells, however, were normal or very nearly normal in appearance in all cases of venous obstruction. In six cases of streptomycin poisoning they were at least in fair condition, perhaps a little pyknotic or shrunken. We can say with confidence that the contribution of the internal hair cells to the CM response is at most quite small compared to that of the external hair cells. In several cases we cannot tell whether the small residue of CM (five to ten per cent of normal maximum) came from good internal hair cells that were present near the electrode, or from scattered or shrunken external hair cells that were found a millimeter or so away. Number 1120 shows that internal hair cells can at least appear normal under the microscope without yielding a CM response, but this case is complicated by abnormal stria and low DC potential.

In summary: *Our evidence strongly supports the hypothesis that the external hair cells are the anatomical locus of the generation of the cochlear microphonic, and that the internal hair cells contribute little or nothing to this response.*

The Summating Potential.

SP is a DC response to acoustic stimulation. The input-output curve of the summating potential is much more variable from animal to animal, and under different conditions in the same animal, than is the response curve of the cochlear microphonic. SP is observed as a displacement of the baseline of the CM in the direction indicating that scala vestibuli has become more negative relative to scala tympani (cf. 9). A full description of the summating potential is in preparation for publication elsewhere. In general, however, *1.* the threshold of detection of SP is 20 to 30 db. above that of CM; *2.* the first segment of the response curve is linear, like CM; *3.* the upper segment at sound pressure levels above 100 db. SPL is relatively stable and continues to rise up to destructive sound pressure levels (see Figs. 7 and 8). The SP response is likely to be larger and more stable in the presence

of slight injury, such as mild anoxia, holes in the cochlea, etc., than in fully normal ears.

For the present analysis only two indices proved simple and stable enough to be useful: the first is the "8 μv threshold," *i.e.* the sound pressure level that just elicits an 8 μv SP response, and the SP voltage at the arbitrary level of 110 db. SPL. The values are given in Table I.

Under an injury by either streptomycin or venous obstruction that reduces CM voltage or raises its threshold the SP remains at or near normal values, and sometimes (*e.g.* 1103, 1097) it may remain fairly prominent when the CM is greatly reduced or almost abolished. The SP follows CM in general, however, in its relation to injury to the organ of Corti and does not, in the crucial experiments (1096 and 1121), follow the resting endocochlear DC potential in its relation to the stria vascularis. It appears clearly to be a response of the organ of Corti to acoustic stimulation.

The class of cell in which pathological changes correlate most closely with depression of SP is the internal hair cell. This cell is rather more resistant to injury by any agent we have used than the external hair cell. SP is absent in all eight cases in which the internal hair cells are absent in Turn I. It is present in all cases but one in which they are present, even when they are described as "fair", "shrunk" or "pyknotic nuclei." The quantitative correlation is good in all but three cases, all of which are in the venous obstruction series. One of these (1104) was complicated by a conductive hearing loss so that SP voltage at 110 db. could not be determined; 1103 (see Fig. 11) showed a small SP, and 1120 showed no SP: both in the presence of "normal" internal hair cells. In 1103 we must postulate either partial anoxia of the animal during the test or covert injury to the internal hair cells. In 1120 (see Fig. 5) there is indirect evidence of poor circulation in the distended capillaries found in stria vascularis and in unusually extensive "hemorrhage and precipitate" in the perilymphatic spaces.

We believe that in spite of these two partial exceptions that require additional, although plausible assumptions, and in

spite of the rather small number of cases, the anatomical source of the negative summating potential is the internal hair cells. A small contribution from some external hair cells is also possible.

Action Potentials.

The normal form and the expected variations in the input-output curve for the N_1 action potential spike in response to a 7000 c.p.s. tone burst will be given more fully elsewhere. Fig. 7 (1117) shows a fair sample of such a curve, however. It rises abruptly from a true threshold response, and it continues to rise, but with varying slope, up to injurious sound pressure levels. Two "humps" in the curve are quite typical, one with its maximum near 60 db. SPL, the other about 100 to 110 db. SPL. Convenient arbitrary indices of the AP curve are: *1.* the SPL at threshold; *2.* the microvolt response at 57 db. SPL and the microvolt response at 110 db. SPL.

The selection of these two arbitrary levels is dictated in large part by our impression that the AP response curve is made up of three components, each generated by a separate "population" of neurons and stimulated by a different group of sensory cells (cf. 11). The low-intensity population, centering at 57 db. SPL, seems to depend quite closely on the presence of a good CM response. On the other hand, expt. 1097 (see Fig. 10) is an excellent example of absence of the low-intensity (CM) population but a nearly normal high-intensity population. The CM response in this case is very small. (Note that the scale is logarithmic.) On the other hand the SP response, which is a less sensitive mechanism, is nearly normal.

We believe that the neurons of the low-level population are those activated by the external hair cells and that the CM is part of the mechanism that activates them. We further believe that the high-level population centering at 100 to 110 db. SPL is the group of neurons activated by the internal hair cells and that SP is part of the mechanism that activates them. This hypothesis is specifically formulated for the response of the basal turn of the cochlea to a 7000 c.p.s. tone burst with a 1 msec rise time, but we believe that in general

the external hair cell and CM are a sensitive low-level mechanism, and the internal hair cells and SP are a less sensitive and more rugged high-level mechanism. This hypothesis incidentally explains how the AP output curve can continue to rise at levels above 105 db. SPL, at which the CM output curve is falling.

The final rise in the AP curve is assisted by the "remote response" described earlier by some of us.[6] A tone burst at levels above about 80 db. SPL evokes not only local movements of the cochlear partition and corresponding CM and SP responses, but also a new mechanical wave that in time-course and amplitude corresponds to the envelope of the tone burst. This is a mechanical detector (demodulator) action. The new mechanical wave activates the apical part of the cochlea that is appropriate to its long wave length, which is determined by the rise-time of the burst. The resulting "remote" action potentials from more apical regions add with the action potentials of the neurons that innervate the first turn. They constitute a third, very-high-level population.

Fig. 12 (1108) in which all hair cells, both inner and outer, were absent in Turn I, but in which pyknotic inner and outer hair cells were both present in Turn II, is a beautiful example of a pure "remote" AP response curve. Notice that no SP and only a doubtful 3 μv CM could be detected in Turn I.

An inference from experiment 1108 is that the detector action that initiates the remote wave does not involve the hair cells. It must be a prior mechanical event, like the process that is responsible for the maximum in the CM response curve.

CONCLUSIONS.

The foregoing observations on cochleas poisoned with streptomycin or depressed by extensive venous congestion, taken in conjunction with our previous knowledge of the electrical potentials of the ear, serve to establish or to support strongly the following conclusions and interpretations:

1. Streptomycin, when it has been absorbed by way of the

round window, causes degeneration first of external, then of internal hair cells, and then of supporting cells of the organ of Corti. The stria vascularis is very little affected.

2. The persistence of a normal positive DC endocochlear potential after streptomycin has destroyed the hair cells eliminates the hair cells as possible generators of the DC potential. (It is not reasonable to attribute a DC potential of +70 mv. or more in scala media in the first turn to generators located in higher turns. The "space constant" established by Misrahy[8] for the scala media is too small: in other words, the basilar membrane and organ of Corti are too leaky electrically to distribute the DC potential very far along the cochlea away from the region of its generation.)

3. Extensive venous congestion, both acute and chronic, affects stria vascularis more quickly and profoundly than the hair cells.

4. The systematic depression of the positive DC endocochlear potential by venous congestion supports the theory that the stria vascularis is the generator of this potential.

5. The effect of venous congestion on the cochlear microphonic, on the summating potential and on the action potentials is variable, depending on the exact degree of injury to various tissues in the neighborhood of the electrodes, particularly the hair cells.

6. The DC endocochlear potential is not necessary for the generation of CM. (It may, however, serve to make the CM response larger or more sensitive.)

7. CM is probably the immediate stimulus to nerve fibers near threshold, but a fairly good AP response is still possible with a very small CM.

8. SP is always present when the AP response is greater than that which can be attributed to the "remote response" of the apical region. SP, as well as CM, is probably an effective stimulus to nerve fibers.

9. Internal hair cells that appear normal under ordinary microscopy may not yield a significant CM response.

10. The negative summating potential is, in general, more resistant to depression by streptomycin or by venous congestion than either CM or AP.

11. Injury by either streptomycin or venous congestion tends to make the SP response function more regular and even to increase the negative SP response above normal. (This may be due to elimination of a very vulnerable positive SP response. A strong positive SP appeared temporarily during a single experiment on acute venous congestion.)

12. The negative SP can still be present in the absence of external hair cells. SP has not been seen when internal hair cells are also absent. This does not exclude the possibility that external hair cells, when present, may *also* generate SP, but the external hair cells probably produce all or nearly all of CM, and the internal hair cells probably produce all or nearly all of negative SP. (The external hair cells are the most probable source of *positive SP,* because both positive SP and the external hair cells are highly vulnerable to injury.)

13. The position of the maximum CM response on the intensity axis remains fixed in spite of injury by either streptomycin or by venous congestion. This strongly indicates that the maximum in the CM response, and probably also "breaks" in the SP input-output curves, are almost certainly controlled by physical (acoustical or mechanical) factors prior to the bending of hairs of the sensory cells.

REFERENCES.

1. BÉKÉSY, G. v.: DC Resting Potentials Inside the Cochlear Partition. *Jour. Acoust. Soc. Amer.*, 24:72-76, 1952.

2. BÉKÉSY, G. v.: Gross Localization of the Place of Origin of the Cochlear Microphonics. *Jour. Acoust. Soc. Amer.*, 24:399-409, 1952.

3. BENSON, R. W., and ELDREDGE, D. H.: Variation in Sound Pressure Produced in Guinea Pig Ears Due to Normal and Abnormal Eardrums. *Jour. Acoust. Soc. Amer.*, 27:373-375, 1955.

4. DAVIS, H.: Biophysics and Physiology of the Inner Ear. *Physiol. Rev.*, 37:1-49, 1957.

5. DAVIS, H.; TASAKI, I.; SMITH, C. A., and DEATHERAGE, B. H.: Cochlear Potentials After Intracochlear Injections and Anoxia. *Fed. Proc.*, 14:112, 1955.

6. Deatherage, B. H.; Davis, H., and Eldredge, D. H.: Physiological Evidence for the Masking of Low Frequencies by High. *Jour. Acoust. Soc. Amer.*, 29:132-137, 1957.

7. Kimura, R., and Perlman, H. B.: Extensive Venous Obstruction of the Labyrinth. *Ann. Otol., Rhinol. and Laryngol.*, 65:332-350, 1956.

8. Misrahy, G.; Shinabarger, E.; Hildreth, K., and Gannon, W.: Bioelectric Studies of the Cochlea. *Fed. Proc.*, 16:88, 1957.

9. Pestalozza, G., and Davis, H.: Electrical Responses of the Guinea Pig Ear to High Audio Frequencies. *Amer. Jour. Physiol.*, 185:595-600, 1956.

10. Rosenblith, W. A.: Electrical Responses from the Auditory Nervous System. *Ann. Otol., Rhinol. and Laryngol.*, 63:839-860, 1954.

11. Schuknecht, H.: Ablation Therapy in the Management of Ménière's Disease. *Acta Oto-Laryngol.*, supp., 132:1-42, 1957.

12. Tasaki, I; Davis, H., and Eldredge, D. H.: Exploration of Cochlear Potentials in Guinea Pig with a Microelectrode. *Jour. Acoust. Soc. Amer.*, 26:765-773, 1954.

13. Tasaki, I.; Davis, H., and Legouix, J. P.: The Space-Time Pattern of the Cochlear Microphonics (Guinea Pig), as Recorded by Differential Electrodes. *Jour. Acoust. Soc. Amer.*, 24:502-519, 1952.

14. Tasaki, I., and Fernandez, C.: Modification of Cochlear Microphonics and Action Potentials by KCl Solutions and by Direct Currents. *Jour. Neurophysiol.*, 15:497-512, 1952.

ACETYLCHOLINESTERASE AND THE OLIVO-COCHLEAR TRACT OF RASMUSSEN. (Abstract).*

Harold F. Schuknecht,

Henry Ford Hospital,

Detroit, Mich.

A high concentration of acetylcholinesterase is found on the nerve fibers and endings of all four rows of hair cells in the cat cochlea. When the olivo-cochlear tract of Rasmussen is cut in the brain stem, the quantity of acetylcholinesterase diminshes greatly, and after several weeks disappears almost entirely.

*The full paper will be published elsewhere.

NEURALLY ISOLATED COCHLEA. (Abstract).*

KARL LOWY
and
ARNOLD A. GERALL,
University of Rochester,
Rochester, New York.

Sectioning of the auditory nerve in the anesthetized cat abolishes or greatly reduces the P1-component of the neural click response. This observation re-opens the problem of the origin of the several parts of the click response. Experiments to determine the nature of these components were undertaken and were reported.

*The full paper will be published elsewhere.

SESSION C.

May 16, 1957.

APPLICATIONS AND LIMITATIONS OF AUDITORY TESTS IN SURGERY OF THE STAPES.

(Abstract).

SAMUEL ROSEN,
Mount Sinai Hospital,
New York, N. Y.,
and
MOE BERGMAN,
Hunter College,
New York, N. Y.

The relative sensitivity thresholds of hearing by air conduction and bone conduction, the so-called air-bone gap, seem to be the most meaningful audiologic criterion for the selection of cases for stapes surgery. In general, the amount of hearing loss does not enter critically into the decision to operate, if there is a significant air-bone gap. Other audiologic measures, such as the discrimination score and tests for recruitment give useful information but, in the presence of a significant air-bone gap do not substantially influence the decision for or against surgery.

A technique for determining the degree of fixation of the stapedial footplate, a quantitative Gelle, was described. It is the author's experience that such tests are useful only if they can definitely establish whether the footplate is or is not fixed at all. While the less rigidly fixed footplate apparently can be more easily *mobilized,* stapes surgery is indicated even in the case of a rigidly fixed stapes, since a fenestra ovalis can be made through the footplate.

Questions were raised about the necessity of maintaining an intact ossicular chain for the surgical removal of the air-

bone gap, and a case was shown where, despite the fracture and removal of the crura, the post-operative hearing was better than normal. It appears that there is not yet a predictable relationship between the pre-operative hearing test findings and the degree of hearing improvement achieved through stapes surgery.

THE ACOUSTIC PROBE AND THE DETERMINATION OF STAPEDIAL FIXATION.

OTTO H. MEURMAN,

University of Helsinki, Finland.

In the February issue of *Acta Otolaryngologica*, 1957, I published a report on the use of an acoustic probe for determination of the degree of stapedial fixation. The present paper is based, in the main, on the same clinical measurements and some additional technical studies.

The work was undertaken to discover the possible value of the acoustic probe in studying the mobility of the ossicular chain prior to mobilization, and in particular to determine whether the stapes is rigidly, or only slightly, fixed to the oval window. The pure tone audiogram mostly provides information as to whether the stapes is mobile or immobile. Audiometric studies, however, do not reveal the degree of fixation.

ACOUSTIC PROPERTIES OF THE PROBE.

For the clinical experiments an acoustic probe was used, consisting of an ordinary ear probe attached to the bone conductor of the audiometer, as illustrated in Fig. 1. The acoustic properties of this probe were measured at the State Technical Research Institute. The purity of tone depends essentially upon the pressure exerted by the probe. If the pressure is small, impurity of tone results. A thin probe vibrates on its own resonance frequencies, which changes very rapidly by loading the probe. The velocity of the probe at

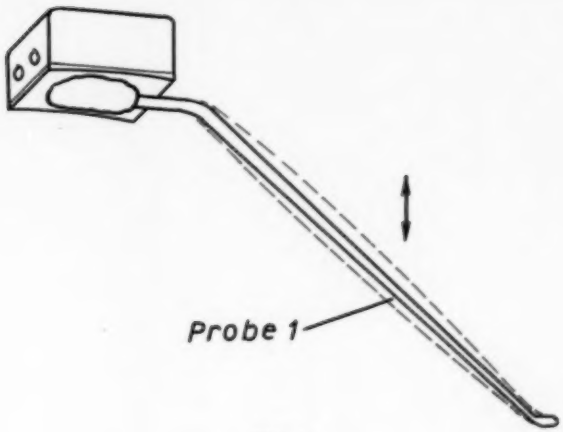

Probe 1

Fig. 1.

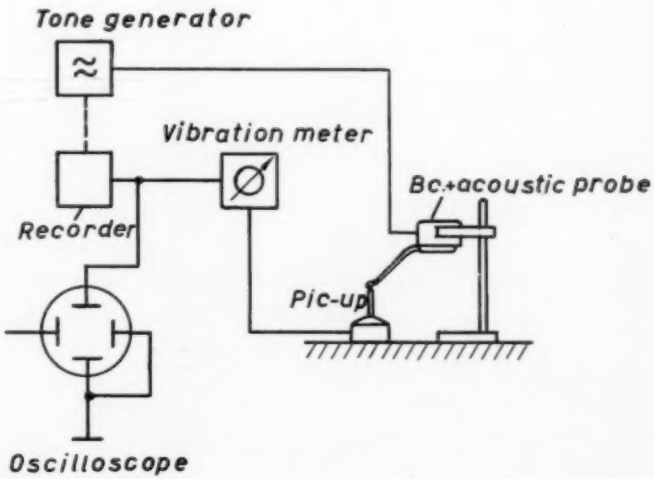

Tone generator
Vibration meter
Recorder
Bc.+acoustic probe
Pic-up
Oscilloscope

Fig. 2.

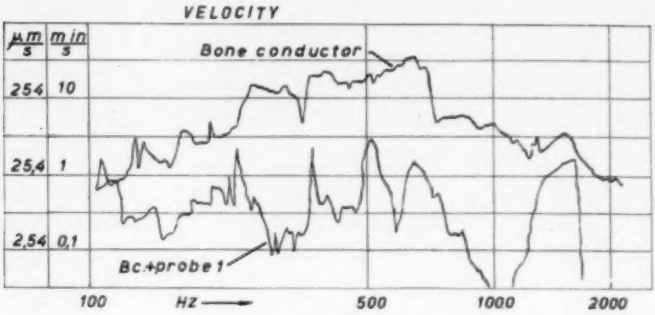

VELOCITY
µm/s
m/s
254
10
25,4
1
2,54
0,1
Bone conductor
Bc.+probe 1
100
HZ
500
1000
2000

Fig. 3.

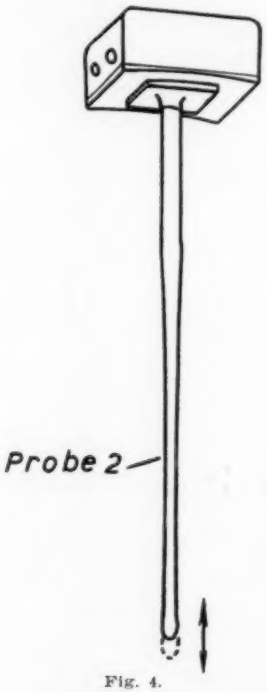

Probe 2

Fig. 4.

various frequencies was tested with the apparatus shown in Fig. 2. The curves in Fig. 3 indicate that the probe, as well as the bone conductor, is most sensitive at about 500 c.p.s., where the velocity is greatest. The amount of energy loss can also be seen from the curves when comparing the velocity coming through the probe with the velocity of the bone conductor itself. Energy transmission is limited to about 0.1 per cent. The second probe (see Fig. 4), has a different con-

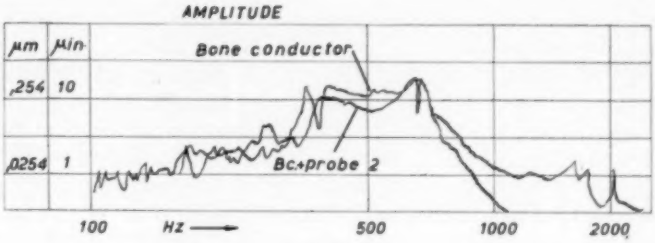


Fig. 5.

struction: the probe itself is thicker than the first and it is attached at right angle to the bone conductor. Vibration here occurs along the length of the probe, transverse vibration being largely eliminated. Sound transmission is thus entirely altered. As shown in Fig. 5, there is scarcely any energy loss between the bone conductor and the probe. A probe constructed along these lines must be regarded as certainly the most suitable testing device, and it will be used in my further studies.

CLINICAL OBSERVATIONS.

A threshold curve was determined at 250, 500, 1000 and 2000 c.p.s., by gently touching the handle of the malleus just below the short process, with the acoustic probe. Superficial cocaine anilin anesthesia was used. To preclude error, the threshold values for the same frequencies were tested also by keeping the probe in the meatus without touching the meatal walls, malleus or tympanic membrane. The values thus ob-

tained were always considerably higher than those obtained from the malleus handle.

Normally hearing ears served for control purposes. The average threshold curve obtained for these ears was taken as the zero curve, by which the hearing loss in the ossicle curves was calculated. The series proper is based on 30 otosclerotic ears; 21 of them were operated upon by stapes mobilization after testing with the acoustic probe. In these

TABLE I.

GROUP 1.

Patient No.	AC - Loss 500 c.p.s.	AC - Loss 1000 c.p.s.	OC - Loss 500 c.p.s.	OC - Loss 1000 c.p.s.
1	40	35	0	5
5	60	60	25	30
15	50	45	20	20
16	55	55	30	20
18	50	40	10	20
19	60	60	15	5
Mean			16.5	16.5

GROUP 2.

2	54	60	35	60
3	50	45	40	30
7	60	50	45	55
8	65	65	60	45
9	50	55	30	40
11	60	60	30	35
13	55	55	40	35
Mean			40	42.9

Group 1. Stapes fixation slight.
Group 2. Stapes fixation rigid.

cases the preoperative ossicle curves could be compared with the degree of stapedial fixation noted at operation. The following table shows the ordinary air conduction hearing loss and the relative ossicle hearing loss (OC) at 500 and 1000 c.p.s. in two different groups of patients. In the first group the stapes was slightly fixed; in the second it was rigidly fixed.

A study of the table readily shows, for one thing, that in Group 1 the air conduction loss is much greater than the ossicle hearing loss, whereas in Group 2 the difference is far

less. It is also seen that in Group 1 the ossicle curve shows values considerably smaller than in Group 2. In the former group fixation of the stapes was slight. Mostly it was fixed only at the anterior periphery. Mobilization in these cases was easy and successful. In Group 2, on the other hand, the stapes was extremely firmly fixed. In some cases mobilization failed; in some it could be effected only by freeing both the anterior and the posterior periphery.

CONCLUSIONS.

Clinical studies show that threshold audiograms, recorded by acoustic probe direct from the malleus handle, present values differing according to the degree of fixation. If fixation is slight, the ossicle curve gives considerably smaller values than the air conduction audiogram. If the stapes is solidly fixed, the ossicle hearing loss at the various frequencies is distinctly greater than in cases with slight fixation. In the presence of only slight fixation, the direct contact between the vibrating probe and the ossicular chain causes vibration also in the stapes footplate, or at least in a part of it. With an increase in the degree of fixation, the stapes functions more and more as a part of the labyrinthine wall, and its vibration is reduced in accordance. From this it follows that, at least in certain cases, it is possible to determine the degree of fixation of the stapes preoperatively by means of an acoustic probe.

DIRECT AUDIOMETRY IN AUDIOSURGERY.*

(Abstract).

FRANCISCO ANTOLI-CANDELA,
FERNANDO ANTIOLI-CANDELA,
F. OLAIZOLA
and
L. SAFT,
Madrid, Spain.

Modern audiosurgery deals not only with fenestration and

*This abstract was received and appeared on the official program, but the authors were not able to be present to present the full paper.

stapedioclasia for otosclerosis, but also with every technique for the recuperation of any deafness caused by congenital defects in the sound transmission, traumatic lesions and suppurated chronic processes of same, and its healing sequels.

With a view to reaching a more exact preoperative diagnosis, and providing beforehand the most adequate surgical technique, we usually carry out a direct audiometry by means of a plastic acoustic conductor connected to a micro-audiometer and placed either on the pathologic formations of the tympanic cavity or on the basic points of the sound conduction, thus enabling us to find out the highest or lowest integrity of the tympano-ossicular apparatus and the permeability of the windows.

It also allows us, when performing a mobilization operation, to know more definitely when we obtain a total or partial mobilization of the stapes and the function of the round window.

Similar findings can be achieved if we encounter remains of ossicles in the audiosurgery of atresias.

NEW APPROACH TO THE INCUDO-STAPEDIAL REGION FOR MOBILIZATION OF THE STAPES.* (Abstract).

LUIS GARCIA-IBANEZ,

Valencia, Spain.

Our experience in mobilizing the stapes according to the approach of Rosen has not been good. With that approach and working on the neck of the stapes, only one out of three patients who were operated upon achieved a result that was lasting and socially useful.

Our attempts to work directly on the footplate of the stapes showed that it is difficult, with Rosen's approach, to achieve good exposure and visualization of the incudo-stapedial

*This abstract was received and appeared on the official program, but the author was not able to be present to present the full paper.

region. This is because the visual direction of gaze is oblique relative to the internal surface of the tympanic cavity, due to its characteristic inclination. This tangential visualization also encounters the obstacles of the short process of the incus and both processes of the stapes.

We have attempted to achieve a better visualization of the footplate by means of a lengthened, preaural incision in the anterior wall of the auditory canal just at the edge of the tympanic bone, which at this place forms the bony portion of the canal. This permits the uncovering of the edge of the tympanic bone. The lifting of the skin that covers both it and the capsure of the temporo-maxillary articulation further permits us to extirpate small or large portions of the bone with the drill. Now we can adjust the visual angle so that it may be perpendicular to the footplate of the stapes.

Further extirpation and laying back of skin permits enlargement of the visualized space so that the footplate may be projected frontally in the center of the visual field and may thus be manipulated more safely and under better control.

THE CARHART-SHAMBAUGH FORMULA USED FOR STAPES MOBILIZATION PREDICTION.

FREDERICK R. GUILFORD
and
C. OLAF HAUG,
Houston, Texas.

It has been generally believed and emphasized by Rosen,[1] Kos,[2] Sheer,[3] and others, that it appears impossible at this time to establish audiometrically a workable prediction of the result to be expected from the stapes mobilization operation. It is pointed out that variations in oval window pathology resulting in stapes fixation in large part determine the feasibility of mobilization, and that the nature and extent of this fixation cannot be ascertained short of exploratory surgery.

We realize that the otosclerotic focus may result in varying degrees of stapes ankylosis, sometimes preventing successful mobilization, and the difficulties involved in establishing an accurate formula comparable to the fenestration prediction; however, we felt that a study of one of the fenestration prodiction formulas on a series of stapes mobilization cases employing the same selection technique by the same audiologist-otologist team, might provide a guide as to what could be expected from the procedure.

TABLE I.

Stapes Mobilization—Closing the Air-Bone Gap.

	Actual Result	CS Prediction
Average Post-Op Level	27.9 db.	28.3 db.
Average Pre-Op Bone Cond.	13.3 db.	13.3 db.
	14.6 db.	15.0 db.

TABLE II.

Stapes Mobilization—Closing the Air-Bone Gap.
Based on Series of 100 Cases.

10 Cases—Better than bone conduction avg. by	+4.5 db.
2 Cases—Same as bone conduction	– 0 db.
81 Cases—Poorer than bone conduction by	–14.66 db.
7 Cases—Failures.	
100	
Total Difference, 100 Cases	–14.6 db.

From a study of 100 consecutive fenestrations in a series of 500 cases, the Carhart-Shambaugh[4] formula was found to predict the post-operative result with a high degree of accuracy. We have continued to use this formula in all preoperative audiological evaluations, even though the stapes mobilization was to be the elected procedure. A previous evaluation of an earlier series of the first 100 stapes operations suggested the possibility of the C-S formula for predictive purposes.

It has been our experience that stapes mobilization attempts do not usually restore complete efficiency of the conductive mechanism, as would be demonstrated by closure

of the bone-air gap. As shown by the results in Tables I and II, the average amount by which this closure fails in our series is 14.6 db.

This figure compares very favorably with the differential of 15 db. between the post-operative air average and the pre-operative bone average (or 25 db. from the corrected bone average), which serves as a basis for the C-S formula. In the fenestration prediction it represents the irreversible residual of hearing deficit resulting from surgical disruption of the impedence matching ossicular chain mechanism.

TABLE III.

Stapes Mobilization—Chances for Closing Air-Bone Gap. Based on Series of 100 Cases.

% Closed Air-Bone Gap	12.9
% Closed Air-Bone Gap within 5 db.	18

Table III further illustrates the small percentage of possibility of closing the bone-air gap completely or within 5 db. This differential between the post-operative air result and the pre-operative bone, which in our series has been so much like that found in the fenestration situation, requires further study to establish an explanation. After stapes mobilization, of course, the ossicular mechanism has not been sacrificed and, theoretically, if complete mobilization is successful, hearing should be restored to the level of the pre-operative bone conduction threshold.

Middle ear pathological changes present at the time of operation, or following mobilization attempts, probably prevent this theoretical bone-air closure. These changes might include: friction in movements of the stapedial foot plate, caused by bony spicules and other irregularities of outline following lysis of the stapes; stretching of the incudo-stapedial or incudo-malleolar joint; fibrosis of the annular ligament; tension changes in tympanic membrane and incomplete mobilization of the stapes footplate. We must bear in mind that mechanical mobility of the stapes does not necessarily insure adequate physiological mobility of the inner ear fluids.

It is our feeling, that given a standardization of pre-operative evaluation involving the same audiologist, and of surgical procedure by the same operator, the main remaining variable in the application of C-S formula is the nature and extent of the otosclerotic pathology which would permit or prevent mobilization of the stapes.

From an evaluation of the distribution of mobilization failures in an adequate series, one may be able to predict the percentage of cases which will defy successful mobilization, because of the anatomy and extent of otosclerotic pathology. Then, eliminating this group and leaving those who can be mobilized, one may predict, using an audiometric formula such as the C-S, what the expected level will be.

In order to investigate this possibility the most recent 100 cases of stapes mobilization, in which one month and four months' post-operative results were available, were studied as to the correlation of post-operative result and C-S prediction. The most recent 100 were chosen in order to eliminate the variable of changing surgical technique, and to take advantage of the factor of surgical experience.

The surgical technique for stapes mobilization used in this series of cases was a variation of the original Rosen[5] technique, as modified by House.[6] Mobilization maneuvers are made through the capitulum of the stapes with curved Rosen explorers. If this maneuver does not result in mobilization, attempts are made to mobilize the stapes via the footplate, as described by Rosen. Fenestration of the footplate was not attempted in this series. Surgical audiometry, adapted from the Goodhill[7] technique, was used in all cases.

We chose these cases with both one month and four-month post-operative results because we felt that it was important to establish the degree of stability of the results used in this study. Table IV shows that the average change from first post-operative test to second was only 1.34 db.

This selection of necessity covered a large group of cases, because, although every patient was contacted for retesting, many failed to return for one of these tests, or went on to

have the fenestration before the four months' post-operative test was done.

While we are aware that our selection has eliminated some unfavorable results, it has also eliminated an equal number of favorable results, as illustrated in Table V.

Table VI shows that the sample used was typical of the

TABLE IV.

Stapes Mobilization—Comparison 1st Post-Operative Test, 1 to 2 Months Following Mobilization, with 2nd Post-Operative Test; 3 to 4 Months Following Mobilization.
Based on Series of 100 Cases.

1st Post-Operative Average Level	27.94 db.
2nd Post-Operative Average Level	29.28 db.
Total Average Difference	1.34 db.
1st Post-Operative Average Gain	20.56 db.
2nd Post-Operative Average Gain	19.22 db.
Total Average Difference	1.34 db.

TABLE V.

Stapes Mobilization—Post-Operative Results—96 Eliminated Cases.

More than 10 db. gain and better than 35 db. level. Good.	35-40 db. level and more than 10 db. gain or 25-35 db. level and 6-10 db. gain. Partial.	Less than 11 db. gain and poorer than 35 db. level; or less than 6 db. gain, any level. Bad.
31 Cases	14 Cases	33 Cases
23.09 db. level 22.7 db. gain	35.5 db. level 12.21 db. gain	51 db. level 1.97 db. gain

range of pre-operative levels ordinarily encountered in otosclerotic patients.

Our average post-operative stapes level, as compared to the average Carhart-Shambaugh fenestration predicted level, is shown in Table VII. The levels of 27.94 db. and 28.39 db. represent an average difference of only .39 db. In Table VIII the range of variance from perfect prediction is plotted. It reveals that 48 per cent of the cases were either exactly predicted or were within 5 db. of perfection; 76 per cent of

TABLE VI.

Stapes Mobilization—Range of Pre-Operative Level.
Based on Series of 100 Cases.

76-80—1	1
71-75—11	2
66-70—11	2
61-65—11111 11	7
56-60—11111 11111 11	12
51-55—11111 11111 11111 11	17
46-50—11111 11111 111	13
41-45—11111 11111 11111 11111	20
36-40—11111 11111 11111 1	16
31-35—11111 111	8
26-30—11	2
	100

TABLE VII.

Stapes Mobilization Post-Operative Results Compared to Carhart-Shambaugh Predicted Results.
100 Case Series.
Results based on 1st Post-Operative Test, 1 to 2 Months After Mobilization.

Average Pre-Operative Level	48.50 db.
Our Average Post-Operative Level	27.94 db.
CS Average Predicted Level	28.33 db.
Total Average Difference	.39 db.
Our Average Post-Operative Gain	20.56 db.
CS Average Predicted Gain	20.17 db.
Total Average Difference	.39 db.

TABLE VIII.

Stapes Mobilization.
Based on Series of 100 Cases.

Better Than CS Pred. Level by—		
21 db.—111	3	
11-20 db.—11111 11111 1111	14	
6-11 db.—11111 11111 111	13	
1- 5 db.—11111 11111 11111 11111 111	23	48%
Same as CS Pred. Level—11111 11111 1	11	
Poorer than CS Pred. Level by—		
1- 5 db.—11111 11111 1	11	76%
6-10 db.—11111 11111 111	13	
11-20 db.—111	4	
21 db.—1	1	
Surgical Failures—11111 11	7	
	100	

our cases were within 10 db. of the Carhart-Shambaugh predicted level. Range of variance is further depicted in Table IX; 53 results were better than the C-S predicted level by 9.3 db., and 29 were poorer than the C-S level by 7.7 db., making a total of 82 cases that differed from the exact fulfillment of the prediction by an average of 8.3 db. There

TABLE IX.

Stapes Mobilization—Post-Operative Levels Compared to Carhart-Shambaugh Predicted Levels Based on Results of 100 Cases.

No. Cases	
53—Better Than CS Predicted Level by	9.3 db.
29—Poorer Than CS Predicted Level by	7.72 db.
82—Differ from CS Predicted Level by	8.3 db.
11—Same Level as CS Predicted Level	0 db.
7—Failures.	
100—Total Cases.	

TABLE X.

Stapes Mobilization.

Predicted Poor Result. Less 11 db. gain and Poorer 35 db. level, any level.		Good Prediction. More 10 db. gain and Better 35 db. Level, or More 30 db. gain any level.	
Fulfilled	Not Fulfilled	Fulfilled	Not Fulfilled
5 Cases	6 Cases	60 Cases	10 Cases.
11 Cases.		70 Cases.	

were 11 who obtained a level identical to the prediction, and 7 surgical failures. Twelve cases had a failure result by our standard of less than 11 db. gain, together with poorer than 35 db. level, or less than 6 db. gain any level; however, five of these cases were predicted to be failures, and were classified as selection failures and included in the predictive comparison. On the other hand, seven had a good prediction and actually achieved a failure, and these were classified as true surgical failures and eliminated from the comparison.

Table X demonstrates the success of the C-S formula in

predicting the extremes of good and poor results. Of the 11 cases predicted for a very poor result, five were fulfilled and six not fulfilled. Of the 70 cases predicted for a good result, 60 cases were fulfilled and 10 cases not fulfilled.

In summary, it has been shown that the Carhart-Shambaugh formula for predicting fenestration results may, according to this series of cases, have value in predicting the stapes mobilization result. It was shown that the average difference in predicted and actual levels was low, and range of variance relatively small. It was demonstrated further that the average amount by which our results fail to close the bone-air gap is almost identical to that which is expected in using the Carhart-Shambaugh formula for fenestration prediction. The number of cases achieving levels close to the prediction was great, and the formula's ability to predict good results appeared significant.

It is emphasized that further study is necessary to explain all the reasons for failure to close the bone-air gap in the majority of cases. Limitations in the formula's use have been illustrated by the fact that although the average difference in predicted and actual result was small, since better and poorer prediction results tend to cancel each other, individual cases may be considerably off from the prediction; also, though the formula had considerable success in predicting good results, it was only 45 per cent effective in predicting very poor results (5 out of 11).

Although much more study is necessary before we will achieve a stapes formula with the predictive ability of the fenestration formulas, we feel that the Carhart-Shambaugh formula, if cautiously interpreted, will serve as a better guide to the expected result in the mobilized cases than is otherwise now available.

BIBLIOGRAPHY.

1. Rosen, S.: Symposium on Stapes Mobilization. The Laryngoscope, 66, 729-783, July, 1956.

2. Kos, C.: Symposium: Stapes Mobilization. The Laryngoscope, 66:729-783, July, 1956.

3. Scheer, Alan A.: Evaluation of Transtympanic Mobilization of

Stapes as a Treatment for Otosclerosis. *E.E.N.T. Monthly*, 34:569-574, Sept., 1955.

4. SHAMBAUGH, G. E., JR., and CARHART, R.: Contributions of Audiology to Fenestration Surgery. *Arch. Otolaryngol.*, 54:699, 1951.

5. ROSEN, S.: Mobilization of the Stapes to Restore Hearing in Otosclerosis. *N. Y. St. Jour. Med.*, 2650-2653, 1953.

6. HOUSE, HOWARD: Personal Communication.

7. GOODHILL, V.: Surgical Audiometry in Stapedolysis. *Arch. Otolaryngol.*, 62:504-508, 1955.

AUDITORY FATIGUE AND OCCUPATIONAL DEAFNESS.*

H. A. E. VAN DISHOECK,
and
A. SPOOR,
University of Leyden,
The Netherlands.

INTRODUCTION.

In the last several years studies dealing with the effect of noise on man were published by the co-workers of the Leyden Otological Clinic. J. van Gool studied the methods of recording auditory fatigue and the dependence of the pattern of the acoustic dip on frequency, intensity and exposure time of the stimulating tone. H. A. van Leeuwen reported on the occurrence of noise trauma and noise deafness in groups of employees in different kinds of factories. C. Rol, together with A. Spoor, gave the results of laboratory studies on the traumatizing effect of steady and nonsteady sounds. A difference between the Leyden investigations, and all previous studies such as the well-known works of Temkin (1933), Børge Larsen (1939), Rosenblith (1942), Kristensen (1946), Kryter (1950), and the report of the American Standards Association Z 24-X-2 is the use of continuous-frequency audiometry instead of octave audiometry.

*From the Oto-Rhino-Laryngological Department of the University of Leyden, Holland (head: H. A. E. van Dishoeck).

CONTINUOUS AUDIOMETRY.

In continuous audiometry a sweep tone is presented to the patient at a certain intensity level. The patient's hearing threshold is noted by asking him to indicate at which frequency he begins to hear the sweep tone, and at which frequency the hearing ends. By doing this at different intensity levels a complete and very accurate audiogram can be made in a short time. The procedure is the contrary of octave audiometry, in which the threshold of some selected frequencies is determined by means of a changing intensity. Thus greater and more exact information can be obtained.

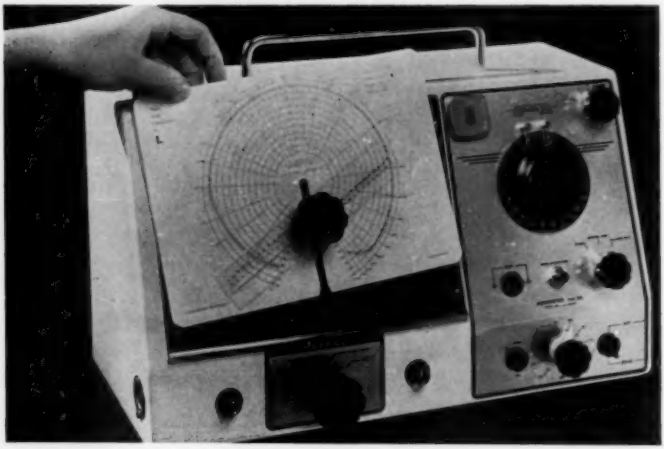


Fig. 1. Continuous audiometer with direct recording on the audiocard. (Peekel).

The Peekel-audiometer is specially built for continuous audiometry. Compensation is made for the low and high frequencies, so that the zero-level of the instrument coincides with the threshold level for normal young persons. As from 200-8000 c/s in one sweep any level above thresholds can be tested, this arrangement makes the audiometer ideal for screening. If somewhere a hearing loss, such as a beginning acoustic trauma, exists, the localization of this loss will always

be detected, and with some additional measurements on higher levels the exact pattern can be established.

EXACTNESS OF CONTINUOUS AUDIOMETRY AS COMPARED TO OCTAVE AUDIOMETRY.

Apart from the greater information obtained by continuous audiometry, because the exact pattern of a hearing loss is established, the reproduceability also proved to be superior. Alternately three continuous and three octave audiograms were made of the same test person by van der Waal. A great number of these audiograms were studied statistically. The mean average standard deviation in continuous audiometry proved to be 2.41 db., whereas in octave audiometry, 3.68 db.

BACKGROUND NOISE.

The great practical difficulty in obtaining reliable audiograms in factories is the fact that the quietest room is not comparable to a specially built silent room of an acoustic laboratory; however, bringing the factory workers to the institute is generally not convenient. The examiner, working in a relatively noisy surrounding and finding threshold anomalies, must know what part of the measured hearing loss is due to masking by background noise and what part can be attributed to an acoustic trauma.

An investigation of this problem was made by Spoor. In a sound-treated room, audiograms of persons with normal hearing were taken during exposure to different kinds and levels of noise, *e.g.* white noise, industrial noise and octave band noise. The audiometer was checked by using as a reference value the average continuous audiogram of 20 young persons without evidence or history of hearing damage. It appeared from these investigations, in confirmation of earlier publications, that the audiogram of a normal test person is already disturbed by a noise level of 20 db. and more. Using telephones with rubber cushions, giving 10-20 db. isolation *the acceptable noise level* in the room will be 30-40 db. In most circumstances this level can be realized for high tones by simple noise isolation measures, but it is often impossible for the low tones; consequently for low tones the unavoidable

noise level is mostly higher than is acceptable, and thus an inaccuracy of the audiogram must be reckoned with. By establishing the threshold shift of normal persons under these unfavorable circumstances, a reference value for pathological audiograms, taken under the same circumstances, can be obtained. Hearing losses as large as, or smaller than, this threshold shift cannot be measured. For instance, when a dip of 10 db. is measured, and the threshold shift by background masking in this frequency region is 15 db., then the real dip, when measured in a silent room, would be 25 db. The average audiogram of 20 normal young persons, taken in a relatively silent factory room, having a background noise level of less than 40 db. in each octave band, proved to be for the higher tones in good accordance with the curves of Dadson and King, and Wheeler and Dickson. Under 100 c/s an apparent hearing loss, due to low-pitched background noise was present. In experienced listeners the differences between the readings in a silent room and in the presence of a background noise are smaller than in unexperienced listeners.

EXPERIMENTS WITH SINGLE PURE TONES.

Auditory fatigue is characterized by a temporary threshold rise of a typical pattern—a tonal dip. The threshold at the frequency of the stimulating tone itself is not, or only slightly disturbed. The maximum of the dip is situated approximately one-half octave above this frequency. For dips caused by 1000 c/s and higher tones the slope of the dip on the side of the lower frequencies is steep and on the other side much more gradually, showing some small depressions. The steepness, and thus the place of the maximum of the dip in regard to the stimulating frequency, depends on the size of the dip. Small dips are bowl-shaped with their maximum on a higher frequency. The extent of the dip depends on the sensitivity of the testees and the traumatizing power of the stimulus. In steady pure tones this power depends on intensity, frequency and up to a certain limit on the exposure time. Thus the same small dip can be obtained when using a certain tone of high intensity during a short time, or of low intensity during a long time. Lengthening of the ex-

posure time, however, over 15 minutes does not increase the extent of the dip. The conclusion from this fact is that in longer exposure, auditory fatigue depends upon intensity and frequency alone; thus in factory workers the resulting trauma might be limited by the existing noise level, and might not depend on the number of years of exposure. Pure tones and noise bands around and above 2000 c/s possess a greater traumatizing effect than lower frequencies of the same intensity above threshold.

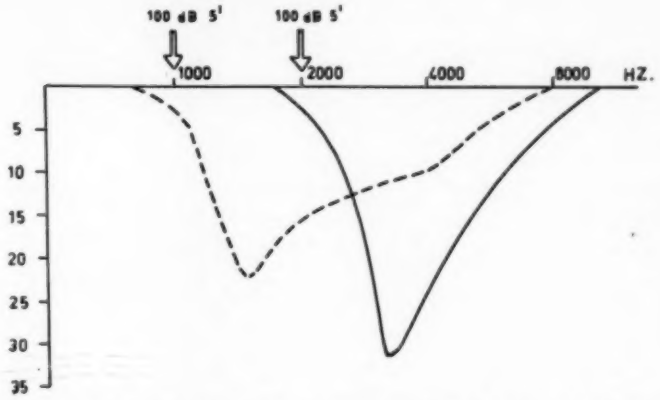


Fig. 2. Pure tone dips caused by 1000 c/s and 2000 c/s tones of the same intensity and duration.

From these general properties of the acoustic dip, one may conclude that the notion of a special sensitivity of the C_5 region is erroneous. Consequently no theories trying to explain the C_5 dip are needed. The origin of the illusion of a preference of the region around 4096 is clearly the result of the measuring at this frequency in octave audiometry. Accordingly, the maxima of industrial noise traumata can be located at any frequencies between 2000 and 7000 c/s.

A THEORY ON THE ORIGIN OF THE ACOUSTIC FATIGUE AND TRAUMA.

Perhaps the tonal dip might be explained by the following

theory: The stimulation pattern of the basilar membrane by a pure tone, as established by its masking effect, betrays what happens when acquiring a tonal dip. The part around the stimulating frequency is strongly but adequately stimulated. Clearly this physiological, adequate stimulation results in the hearing of a pure tone, and none, or only a small

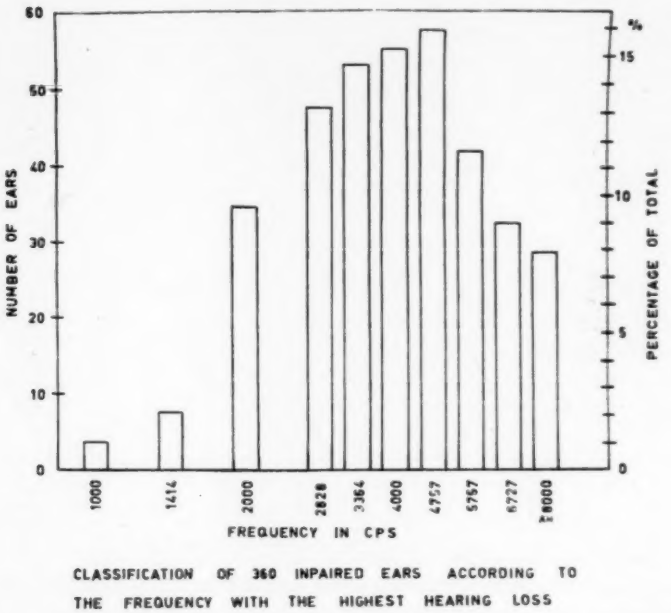


Fig. 3. Classification of 360 noise traumata according to the frequency with the highest hearing loss.

threshold rising, can be detected afterwards; moreover, the perstimulatory loudness adaptation is present around this stimulating frequency, and not above or below.

As is demonstrated by von Békésy the basilar membrane between the stimulating frequency and the windows suffers distortion due to swinging. According to the calculations of

Ranke and Reboul, the maximum of this distortion is situated about one-half octave above the stimulating frequency, thus on the place of the maximum of the dip. We assume that this part is damaged by such an inadequate stimulation. The amplitude of the swinging is higher nearer to the stimulating frequency, which fact agrees with the slope of the curve and the place of the maximum of the dip. Notwithstanding the relatively large movements, this inadequate form of stimulation causes no hearing sensation. Perhaps the fact that in high intensities a pure tone sounds more or less impure, is due to this distortion.

Frequent repetition of the reversible auditory fatigue, without sufficiently long period of recuperation, results in an irreversible lesion of the organ of Corti; that is, in an acoustic trauma.

FATIGUE CAUSED BY PURE TONES OR BY A WHITE NOISE.

The effect of stimulation by two pure tones proved to be the combination of two separate dips. Consequently it might be possible that, if an employee is traumatized by a high pitched noise and he is transferred to a department having a low pitched noise, an additional trauma for lower frequencies will result. Fatigue caused by one-third octave bands proved to be similar to the effect of pure tones.

As a white noise is the combination of a great number of frequencies, the resulting trauma will depend upon the sensitivity of the ear and the traumatizing power of the different frequencies. The frequency bands around 2000-3000 c/s are dangerous, because the threshold of the ear is low, and the traumatizing power of these higher tones is great. The trauma of a white noise proved to be a broad bowl shaped dip, extending from about 3000-7000 c/s; consequently, in an industrial noise the energy in the 2000-3000 c/s band is of great importance.

EXPERIMENTS WITH PULSE TONES AND PULSATING NOISES.

Rol and Spoor studied the traumatizing power of non-steady sounds as compared to the better known traumatizing

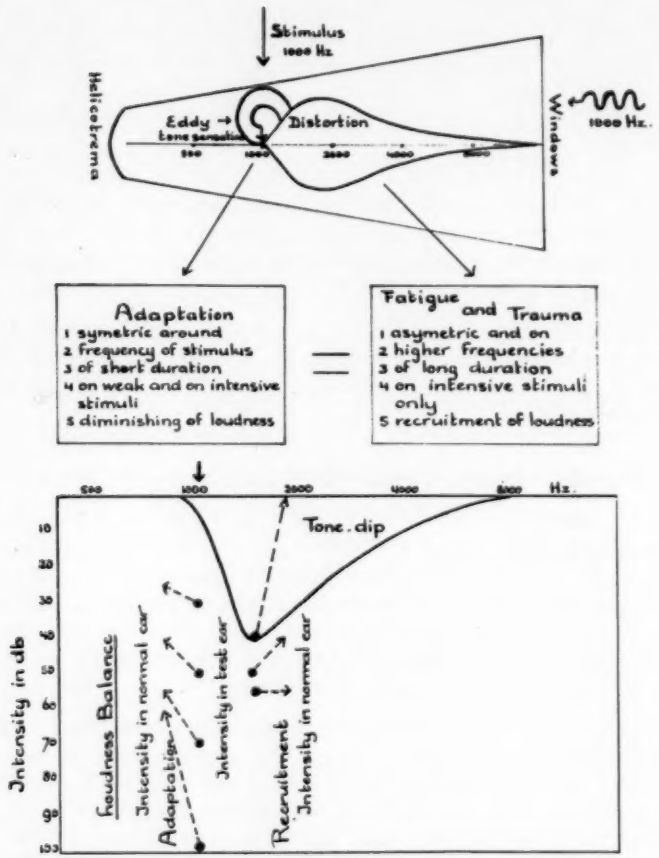


Fig. 4. Schematic representation of cochlear mechanisms underlying adaptation and fatigue.

power of steady sounds. Intermittent pure tones, an intermittent white noise, synthetically constructed sounds imitating pulsing factory noises, and the recorded noise of a pneumatic drill, were used. It appeared that a sound-level meter, whether or not combined with band filters, does not give exact values for the traumatizing power of pulsating sounds.

Intermittent stimuli are less traumatizing than continuous stimuli of the same calculated overall intensity.

Increase of the pulse duration, the pulse-rate being constant, causes a greater increase of trauma than would be expected from the corresponding increase of the overall-intensity (see Fig. 5).

In the same way it was established that increase of the pulse-rate, the pulse-duration being constant, has a greater influence on the traumatizing power than on the calculated overall-intensity of the tone (see Fig. 6).

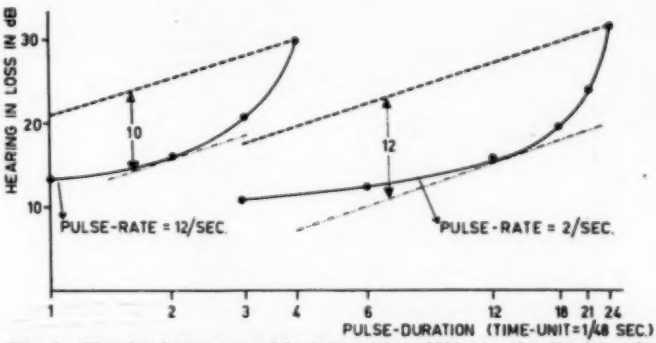


Fig. 5. Hearing losses caused by intermittent 2000 c/s stimuli with pulse-rates of 2 and 12 per sec. The upper-dashed lines indicate the hearing losses corresponding to the continuous stimuli of the same overall-intensities.

The difference between the trauma caused by an intermittent pure tone and that caused by a continuous one of the same calculated overall intensity was maximal; *viz*: 7 to 12 db. for a pulse interval ratio of about 1:1. When keeping the pulse interval ratio constant, it appeared that a pulse-rate of 8 per second is more harmful than one of 2 per sec. If both noise and superimposed peaks are traumatizing, the effect of the total noise proved to be equal to the sum of the traumatizing powers of the components.

The well-known deafness risk criteria are calculated from the threshold curve of the ear and from the greater trauma-

tizing of the high tones. In order to establish this line experimentally Rol determined, in a number of testees for several tones, the minimum stimulation necessary to cause fatigue. This empirically obtained curve differs, especially for the so important 2000-3000 c/s band, from the theoretically calculated curves (see Fig. 7). It is known that the social validity for hearing of speech depends for a great deal on the range above 1500 c/s. Thus, especially, excess of

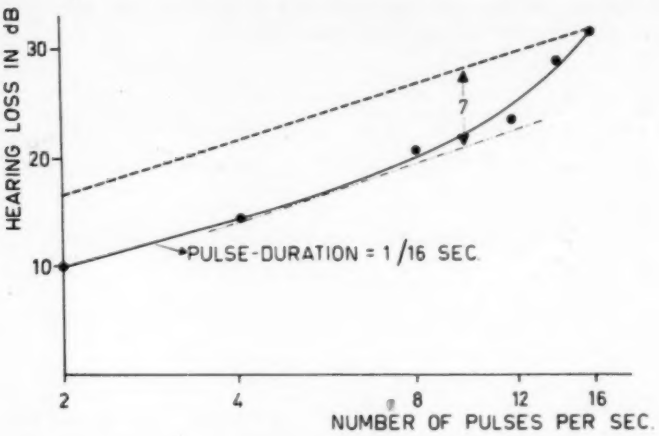


Fig. 6. Hearing losses caused by intermittent 2000 c/s stimuli with a pulse-duration of $\frac{1}{16}$-second. The upper line indicates the hearing losses corresponding to the continuous stimuli of the same overall-intensities.

energy in the 1000-3000 c/s band will endanger the speech intelligibility.

An attempt was made to establish this tone-sensitivity individually and in a quick and easy way. Departing from the supposition that a tone becomes uncomfortable when the traumatizing threshold is reached, we asked our testees to indicate at which intensity a pure tone acquires an impure and sharp character. Indeed, the curve in this way obtained, the tolerance curve, proved to be a good criterion for the individual sensitivity of noise (see Fig. 7).

INDIVIDUAL DIFFERENCES IN SENSITIVITY TO NOISE.

It is a fact of common experience that among workers who are exposed to the same noise intensities, considerable differences in acoustic trauma exists. Even the difference between the left and right ear of an employee can be very great.

As we mentioned above, the tolerance curve (see Fig. 7) may give us an idea of the traumatizing threshold of dif-

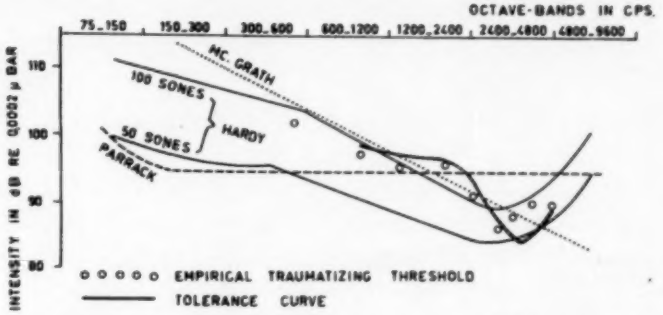


Fig. 7. Deafness risk criteria, empirical traumatizing threshold and tolerance curve.

ferent subjects. Usually, however, an above threshold method is employed, either by measuring the dip caused by a pure tone or by a noise. In order to study this problem, van Leeuwen exposed 108 ears to 2800 c/s tone of 100 db. during three minutes. As a tonal dip changes very rapidly in size, the resulting dip was measured by means of continuous audiometry in one minute, beginning one-fourth minute after the cessation of the stimulating tone.

Threshold shift in db.; stimulus: 2800 c/s, 100 db., 3 minutes.	No. of Ears.
0:–2-+2	26—24%
5: 3- 7	27—25%
10: 8-12	33—31%
15:13-17	8— 7%
20:18-22	7— 6%
25:23-27	4— 4%
30:28-32	3— 3%

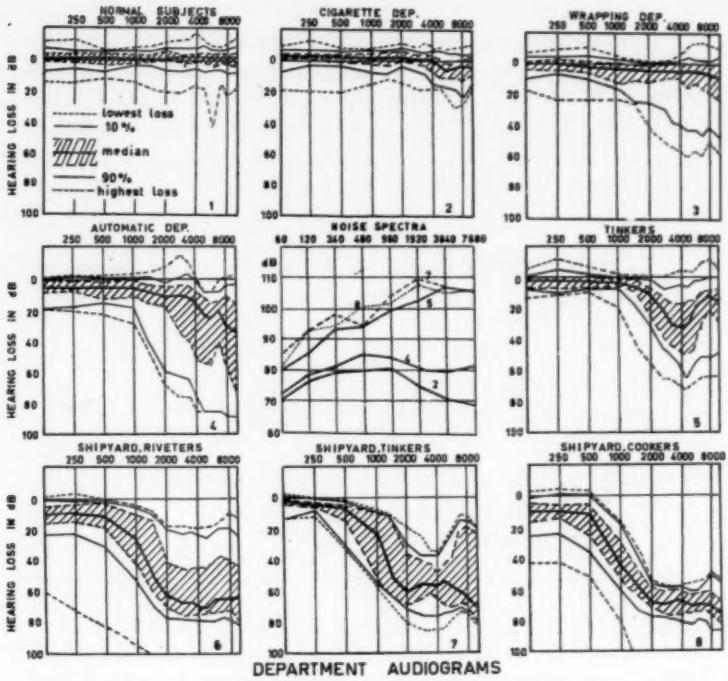


Fig. 8. Combined audiograms of employees in departments with different noise levels.

From these results we may conclude that considerable individual differences in sensitivity exist. Against 24 per cent without threshold shift, 20 per cent proved to have a threshold shift, varying from 15-30 db.

NOISE TRAUMA AMONG FACTORY WORKERS IN DORDRECHT.

In order to study the correlation between industrial noise level and resulting hearing loss, van Leeuwen classified 279

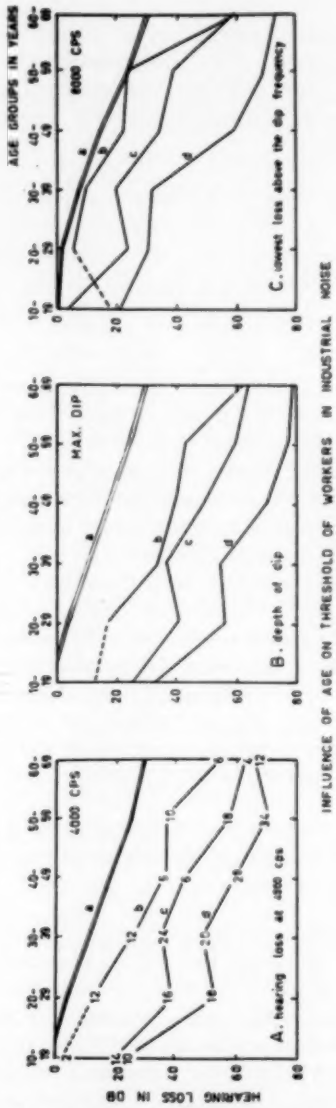

AGE GROUPS IN YEARS
4000 CPS
C. lowest loss above the dip frequency
MAX. DIP
B. depth of dip
4000 CPS
A. hearing loss at 4000 cps
HEARING LOSS IN DB
INFLUENCE OF AGE ON THRESHOLD OF WORKERS IN INDUSTRIAL NOISE
a NORMAL SUBJECTS (BEASLEY)
b PUNCHING DEPARTMENT
c BOILERSHOP
d SHIPYARD

Fig. 9. The influence of age on the hearing of employees in different departments.

employees of ten noisy departments of six different factories on the Isle of Dordrecht. The audiograms of the employees of each department were combined by considering the median curves and checking them statistically with a group of normal controls.

It appeared that these department audiograms could be classified according to the prevailing noise levels. This fact agrees with the above mentioned conclusions, that, generally speaking, the size of the trauma depends mainly on the quality and intensity of the stimulus, and not on the time of exposure to noise.

In comparing the audiograms of newly appointed employees after a day's work, and the next morning, their capacity for recovery could be established. In those who worked some years in a certain department, this recovery was practically absent; moreover, the very important fact was found, that the post-working audiograms of the newly appointed workers resembled closely those of the older employees. This observation suggests that repeated acoustic fatigue results in a permanent trauma of the same size. From this supposition we may expect that in each department the mean audiograms of different age groups, corrected for presbycusis, should present the same degree of trauma, particular for that department. This conception proved, indeed, to be substantially true, only a slight deterioration occurring in the course of the years, which is not accounted for by presbycusis.

From these observations, too, the important fact appeared that the permanent trauma is already acquired after a relatively short exposure time.

Van Leeuwen calculated the average degree of trauma in groups of workers of about the same exposure time to the same noise level, in order to see whether there was an increase in loss in the groups with longer exposure to noise. Since longer exposure and higher age are inevitably linked together the influence of presbycusis was eliminated by correcting the individual audiograms. It appeared that most of the damage is done the first year of exposure to noise.

SUMMARY.

From our studies we may conclude that a certain average hearing loss belongs to a certain noise level. After some months of exposure the amount of hearing loss hardly depends on the exposure time any more, but mainly on the noise level to which the factory worker is exposed. In a relatively short time a trauma is acquired corresponding to the noise level, and thereafter, from this cause the hearing loss does not increase very much. A greater damage may occur when the employee shifts work to a department with a higher noise level or with another pitch. When in course of time presbycusis develops, hearing loss from this cause is added to the existing acoustic trauma. Thus, a noise trauma acquired in youth may be unnoticed, but at a higher age this same trauma may be the cause of a hearing impairment for speech. This consideration may have important legal consequences.

REFERENCES.

BÉKÉSY, G. VON: *Acto Oto-Laryngol.*, 35:411, 1947.

DADSON, R. S., and KING, J. H.: *Jour. Laryngol., Otol.*, 66:366, 1952.

DISHOECK, H. A. E. VAN: *Arch. Otolaryngol.*, 64:216, 1956.

GOOL, J. VAN: Thesis Leyden, 1952.

KRISTENSEN, H. K.: *Acta Oto-Laryngol.*, 34:82, 1946.

KRYTER, K. D.: *Jour. Speech Hear. Dis. Monogr.*, 1:1, 1950.

LARSEN, B.: *Acta Oto-Laryngol.*, Suppl., 36, 1939.

LEEUWEN, H. A. VAN: Thesis, Leyden, 1955.

RANKE, O. F.: *Akust. Ztschr.*, 7:1, 1942.

REBOUL, J. A.: *Phys. Rad.*, Serie 7, Tome 9, No. 5, 1938.

ROL, C.: Thesis, Leyden, 1956.

ROSENBLITH, W. A.: *Jour. Acoust. Soc. Amer.*, 13:220, 1942.

ROSENBLITH, W. A., ET AL.: Report Subcommittee, Z 24-X-2, *Acoust. Soc. Amer.*, 1954.

SPOOR, A.: *Acta Oto-Laryngol.*, 44:386, 1954.

TEMKIN, J.: *Mschr. f. Ohrenhk.* u. L. R. 67:257, 1933.

WAAL, J. VAN DER, and DISHOECK, H. A. E. VAN: *Oto-Rhinol.-Laryngol.*, 17:46, 1955.

WHEELER, L. J., and DICKSON, E. D. D.: *Jour. Laryngol., Otol.*, 66:379, 1952.

TEMPORARY HEARING LOSS AT 4000 C.P.S. AS A FUNCTION OF A THREE-MINUTE EXPOSURE TO A NOISE OF UNIFORM SPECTRUM LEVEL.*

JAMES D. MILLER,

Indiana University,

Bloomington, Ind.

INTDODUCTION.

The results of several experiments indicate[2,3,5,6,7,10] that the form of the function relating temporary hearing loss to the intensity of the exposure stimulus is complex. As the intensity of the exposure stimulus is increased from a low value, temporary hearing loss remains at or near zero. With further increases in intensity, the amount of loss at first positively accelerates, and then increases linearly up to high intensities. At the highest intensities, temporary hearing loss may show no further increase, or it may, in fact, decline, with an increase in the intensity of the exposure stimulus. This experiment was designed to provide further information on this relationship, as well as to help answer the following questions:

1. How does the relationship between temporary hearing loss (THL) and intensity differ from listener to listener?

2. How does this relationship change as the threshold recovers?

It is well-known that wide individual differences in THL are observed when all listeners are exposed to the same stimulus.[1,8,9] Answers to the two questions listed above would enable one to predict how the same individuals would differ

*This paper is part of a thesis submitted in partial fulfillment of the requirements for the Doctor of Philosophy degree in the Department of Psychology, Indiana University. The research was supported in part by the United States Air Force under Contract No. AF 18(600)-571, monitored by the Operational Applications Laboratory, Air Research and Development Command, Bolling Air Force Base, Washington 25, D. C. This is Report No. AFCRC-TN-56-70, ASTIA Document No. AD-110052.

if the intensity of the exposure stimulus were raised or lowered.

METHOD AND PROCEDURE.

Six listeners, ranging in age from 23 to 35 years, served in this experiment. All had normal hearing and all data were collected monaurally, using the right ear.

A PDR-10 earphone, which was encased in a doughnut-type cushion (NAF-48490-1) and mounted on a head band, was used to produce pure tones and noise. The apparatus permitted the measurement of quiet absolute thresholds, the measurement of masked thresholds,* and the exposure of the ear to intense noise. The test tone was generated by an audio oscillator. The output of the oscillator was fed to an electronic switch, two attenuators in tandem, a mixer, a matching transformer, and then it was terminated by the earphone. The output of a noise generator was passed through an attenuator to an amplifier. For the measurement of masked thresholds, the output of this amplifier was fed through a matching transformer to the mixer in the tone circuit. When the noise was to be used as an exposure stimulus, the output of the amplifier was put directly across the earphone. Only high fidelity components were used, and switching devices allowed the experimenter to select the appropriate circuit and to keep all terminals properly loaded.

Each listener was run individually in eight experimental sessions. At each session, the absolute threshold at 4000 c.p.s. was determined, the listener was exposed for three minutes to a noise of uniform spectrum level (60-7000 c.p.s.), and the recovery of the threshold at 4000 c.p.s. was traced for 13¾ minutes. The first determination of a post-exposure threshold was completed 15 seconds after the cessation of the noise. During the remainder of the session, a threshold measure was completed every 30 seconds.

At each session a different level of noise was used. There were eight levels, which ranged in overall intensity from 85 db. to 120 db. (*re* 0.0002 microbar) in steps of five db. These

*The data for the masked thresholds are not reported.

levels are estimates of the SPL under the cushion. The level in a 6-cc coupler would be about 8 db. greater. Each listener was exposed at each of these eight levels. The intensity of the noise was 85 db. at the first session and 120 db. at the last session. The intermediate noise levels were assigned to listeners and sessions by means of a 6x6 latin square which was selected randomly; thus, for Session II to VII and for noise intensities 90 to 115 db., the effects of sessions and intensities were counterbalanced.

The threshold measurements were obtained by the method of adjustment. The listener controlled the intensity of the tone by means of an attenuator which had a range of 60 db. with steps of two decibels; he was instructed to bracket his threshold by manual adjustment of the attenuator. A test tone was presented for a 15-second period. By this method the listener was allowed 15 seconds to adjust the tone to his threshold. At the end of the 15-second period, he reported the attenuator reading at which he could just detect the tone. When repeated measurements were obtained, there was a 15-second period of silence between the successive measurments.

The test tone was interrupted once per second by an electronic switch. It was on for .7 seconds and off for .3 seconds. The rise-fall time was 50 milliseconds. Another attenuator was in tandem with the one that the listener manipulated. From trial to trial the experimenter haphazardly varied the amount of attenuation in the second pad. This was done so that the listener would not be influenced by his settings on previous trials.

Temporary hearing loss (THL) is defined as the difference in decibels between the pre-exposure threshold and the post-exposure threshold. The pre-exposure threshold was defined for each listener as the mean of five determinations of the threshold which were obtained immediately before each exposure. It was desirable to obtain the most reliable measure of the THL produced by each intensity of the noise after various periods of recovery. For this purpose the following variables were defined for each listener at each session. The post-exposure threshold at 15-seconds after the cessation of

the noise was defined as the single measurement, which was completed then. The next two measurements, which were completed 45 and 75 seconds after exposure, were averaged to define the post-exposure threshold at 1 minute after the cessation of the noise. The next four measurements of the threshold were averaged to define the post-exposure thresholds at 2½ minutes after the exposure. The post-exposure threshold at 6 minutes after the exposure was defined as the average of the ten measurements of the threshold obtained between 3¾ and 8¼ minutes after the cessation of the noise. Likewise, the post-exposure threshold at 11 minutes was defined as the average of the ten measurements of the threshold in the interval 8¾ to 13¾ minutes after the exposure.

For each listener, THLs were computed for the different times after exposure by subtracting the pre-exposure threshold from the post-exposure thresholds just defined.

In order to detect and to avoid any cumulative auditory effects of this series of exposures, the following precautions were taken: For a given listener, successive sessions were separated by at least 48 hours. Audiograms (500-8000 c.p.s.) were taken at the beginning of each session. If these thresholds were not within six db. of their values at the previous session, then the session was terminated, and the exposure was delayed 24 hours.

RESULTS.

To check on the possibility that there were effects due to either practice or the repeated exposures to noise, the pre-exposure thresholds for the individual listeners were plotted against sessions. No consistent trends appeared in these curves. Also, the mean THLs for Session II to VII were examined. In each of these means, the six listeners and the six intensities (90-115 db.) were represented, since over this portion of the experiment noise intensities and sessions were counterbalanced over listeners. No trends toward greater or smaller THLs were noted; thus, there was no evidence for changes in THLs due to practice or to the repeated exposures to noise.

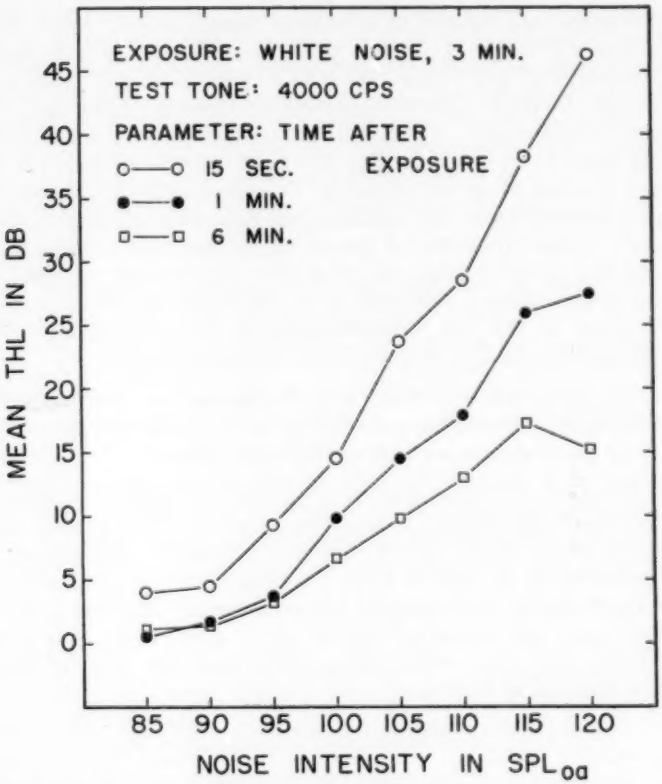


Fig. 1. Temporary hearing loss at 4000 c.p.s. as a function of the intensity of a 3-minute exposure to white noise. The parameter is the time after the cessation of the noise. Each point is the mean of the results for six listeners.

A plot of THL against the intensity of the noise is shown in Fig. 1. Each point is a mean based upon the results from the six listeners. The parameter is time after exposure. The curve for the THLs determined 15 seconds after the exposure is positively accelerated over its early portions, and then it becomes approximately linear with a slope of about one and one-half. Except for the exposure to 120 db., the curves for the THLs one minute and six minutes after the

cessation of the noise roughly parallel the THLs 15 seconds after the exposure. Although not shown in Fig. 1, the curves for the THLs 2½ minutes and 11 minutes after the exposure seem to fit into the family of curves. The mean THL for the different times after exposure is given for each intensity of noise in Table I.

Because of individual differences, the basic relationships among THL, time after exposure, and the intensity of the noise may be distorted by averaging all listeners together. It

TABLE I.

THE MEAN THL AT 4000 C.P.S. FOR DIFFERENT TIMES AFTER THE EXPOSURE AND EACH OVERALL INTENSITY OF THE NOISE.

Time After Exposure	INTENSITY OF THE NOISE							
	85	90	95	100	105	110	115	120
15 seconds	4.0	4.4	9.3	14.5	23.7	28.5	38.3	46.2
1 minute	.6	1.8	3.7	9.8	14.5	17.9	26.0	27.5
2½ minutes	2.2	3.3	5.3	9.0	13.0	15.6	21.0	22.1
6 minutes	1.1	1.6	3.2	6.6	9.8	13.0	17.3	15.2
11 minutes	1.1	1.3	2.6	5.9	8.4	11.9	14.5	12.7

is important to examine the data for the individual listener, and the data are plotted in this way in Fig. 2.

Consider the data for JF shown in panel A. The THLs determined 15 seconds, 1 minute, and 6 minutes after the exposure are shown by the open circles, the filled circles, and the squares respectively. Over their early portions these curves are positively accelerated, and then they become almost linear. That is, once THL has become as large as 15 or 20 db., the remaining portion of the curve can be approximated by a straight line with a slope of about two.

The curves for the other listeners show either an inversion or a leveling off when the highest intensities are reached. This fact will be discussed later. For the present purposes, only the data up to a point of the inversion or leveling off will be considered. This is done by finding for each listener the noise intensity at which the curve in Fig. 2 for THL 6 minutes after the exposure either shows a maximum or levels off. An arrow indicates this intensity for each listener. All THLs for intensities to the right of the arrow are disregarded.

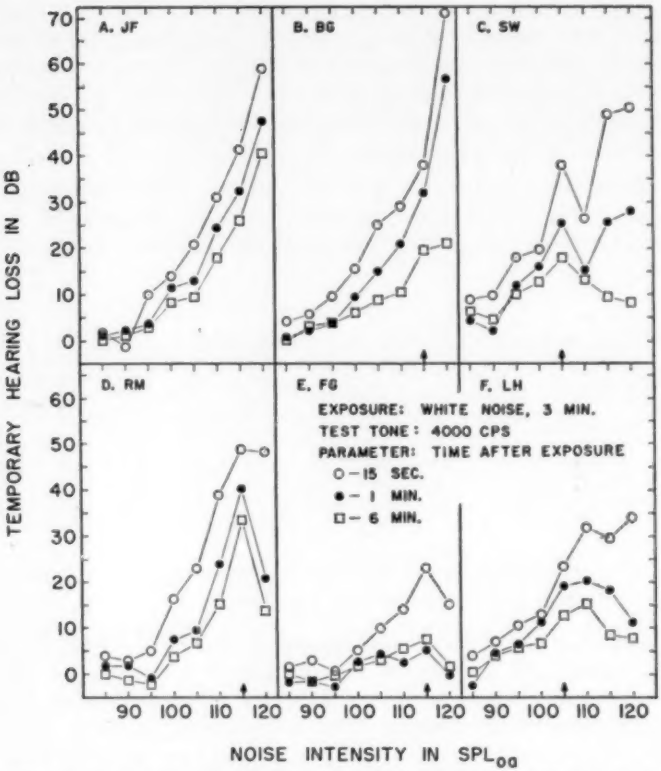


Fig. 2. Curves for individual listeners showing THL at 4000 c.p.s. plotted against the intensity of a 3-minute exposure to white noise. The parameter is the time after the cessation of the noise. The arrow in each panel indicates the intensity above which the 6-minute curve either levels off or declines.

Under these conditions, examination of the data shown in Fig. 2 suggests that for a given listener the curves for the three different times after the exposure can be superposed by shifting their positions along the intensity axis.

In order to facilitate comparisons among the listeners, part of the data of Fig. 2 are replotted in Fig. 3. The upper panel shows the THLs 15 seconds after the exposure, and the lower panel shows the THLs 6 minutes after the ex-

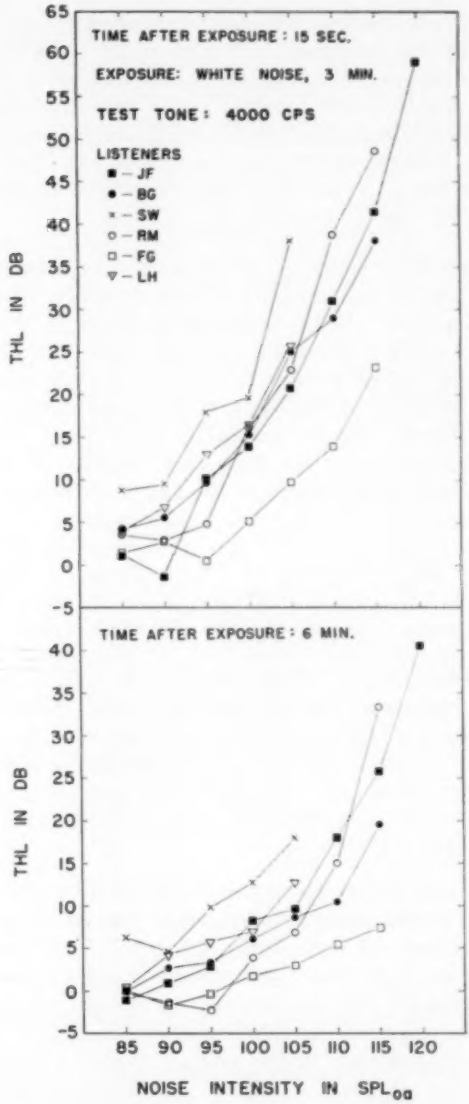


FIG. 3. A comparison of the curves for the individual listeners showing THL at 4000 c.p.s. as a function of the intensity of 3-minute exposure to white noise. The upper panel shows the THLs at 15 seconds after the cessation of the noise. The lower panel shows the THLs at 6 minutes after the cessation of noise. These data were taken from FIG. 2, but only the data up to the point indicated by the arrow are included. See text for explanation.

posure. These curves have been "amputated" as suggested in the previous paragraph. It can be seen that the shapes of the curves are highly similar for all listeners. Inspection of these graphs suggests that the main difference among listeners is the position of the curve along the intensity axis. Of course, this conjecture can be correct only for the THLs up to the intensity at which the inversion or leveling off occurs.

An inversion or sharp deceleration in curves showing THL as a function of the intensity of the exposure stimulus will now be discussed. With an exposure stimulus of about 120 db., Egan,[4] using white noise, and Davis, et al.,[2,3] using pure tones, also found this effect. Davis, et al., add that the intensity at which this "bend-over" occurred varied from listener to listener and for a single listener from day to day. These investigators interpreted their results to mean that some protective mechanisms of the ear, such as a contraction of the intra-aural muscles or a change in the mode of vibration of the stapes, attenuates the effective stimulation of the inner ear. Referring back to Fig. 2 above, if one were to look at only one curve at a time, then the data would fit into the pattern described by Egan and by Davis, et al.

The nature of the change that takes place after THL reaches a maximum can be best illustrated by plotting for each listener the family of recovery curves with the intensity of the noise as a parameter. Fig. 4 shows the data plotted in this way. These are essentially the same data as shown in Fig. 2, but the data for more times after the exposure and fewer intensities are given.

The recovery curves for BG and RM given in panels B and D show an effect present in the data for all listeners with the exception of JF. Recovery from the more intense exposures is so rapid that the recovery curve crosses the recovery curves for the less intense exposures. This crossing of recovery curves can be noted also in the data of SW, FG, and LH. This change in the rate of recovery is quite dramatic, as can be seen by examining the recovery curves before the "bend-over" is reached. The shapes of the re-

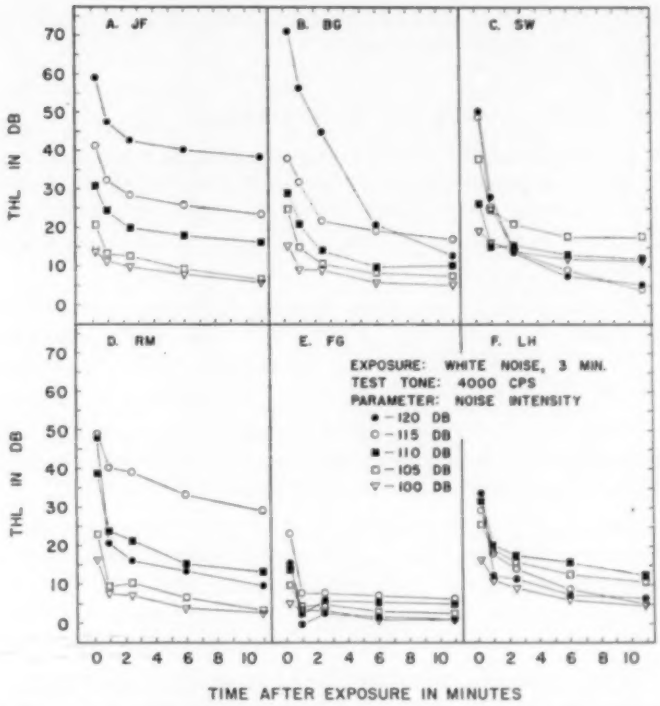


Fig. 4. Recovery of the 4000 c.p.s. threshold after a 3-minute exposure to white noise. Each panel shows the data for an individual listener. The parameter is the over-all intensity of the noise.

covery curves after exposure to the "low" noise intensities are remarkably independent of the noise intensity. Also, listener JF did not show a maximum in his curves for THL plotted against noise intensity (see Fig. 2). The recovery curves for JF in panel A of Fig. 4 all have much the same shape.

The marked change in *rate* of recovery in the results of the experiment reported here cannot be explained readily by assuming that some protective mechanism of the ear simply reduced the effective stimulation of the inner ear during the duration of the exposure. It would seem that such a mechan-

ism would reduce the early THLs as much as the THLs at the later times after the exposure.

SUMMARY OF RESULTS.

1. The shape of the curve relating THL to intensity of the exposure stimulus is nearly the same at different times after the cessation of the noise. The main effect of time after exposure is to shift the position of the curve along the intensity axis.

2. The shape of the curve relating THL to noise intensity is nearly the same for all listeners. The main difference among listeners appears to be the position of the curve along the intensity axis.

3. For THL >15 db., the relationship between THL and noise intensity for the individual listener can be approximated by a straight line with a slope of about two.

The above conclusions are qualified by the fact that a leveling off or maximum in the curves relating THL to noise intensity occurs for most listeners, once the noise intensity becomes as great as 110-120 db. Individuals appear to differ with regard to the intensity at which this effect first occurs.

With regard to individual differences in susceptibility to temporary hearing loss, the following hypothesis is tentatively proposed. A given listener's position in a frequency distribution of THLs, when all listeners are exposed and tested under the same conditions, will depend upon two factors: *1.* the position of his curve (THL against noise intensity) along the intensity axis, and *2.* the intensity at which this curve shows a leveling off or an inversion.

REFERENCES.

1. CHRISTIANSEN, E.: Auditory Fatigue and White Noise. *Acta Otolaryngol.*, 26:99-105, 1956.

2. DAVIS, H., ET AL.: Final Report on Physiological Effects of Exposure to Certain Sounds. OSRD Report No. 889, Dept. of Commerce, Office of Tech. Serv., PB 19786.

3. DAVIS, H., ET AL.: Temporary Deafness Following Exposure to Loud Tones and Noise. *Acta Otolaryngol.*, Suppl. 88:1950.

4. EGAN, J. P.: Personal Communication.

5. HIRSH, I. J., and BILGER, R. C.: Auditory-threshold Recovery After Exposure to Pure Tones. *Jour. Acoust. Soc. Amer.*, 27:1186-1194, 1955.

6. HOOD, J. D.: Studies in Auditory Fatigue and Adaptation. *Acta Otolaryngol.*, Suppl. 92:1-57, 1950.

7. JERGER, J. F.: Recovery Pattern from Auditory Fatigue. *Jour. Speech Hear. Disord.*, 21:39-46, 1956.

8. LIGHTFOOT, C.: Contribution to the Study of Auditory Fatigue. *Jour. Acoust. Soc. Amer.*, 27:356-364, 1955.

9. MILLER, J. D.: "On the Relationship Between Temporary Hearing Loss and Masking." Ph.D. Thesis, Indiana University, 1957.

10. STEVENS, S. S.; EGAN, J. P., ET AL.: "The Effects of Noise on Psychomotor Efficiency; 2. Noise Reduction in Aircraft as Related to Communication, Annoyance, and Aural Injury." OSRD 274, Report MHR-4 Psycho-Acoustic Laboratory, Harvard Univ., Dec. 1, 1941.

CLINICAL BONE CONDUCTION AUDIOMETRY AT THE FRONTAL BONE.

(Abstract.)*

R. F. NAUNTON,

University of Chicago Clinics,

Chicago, Ill.

Our interest in clinical bone conduction audiometry stems directly from early tuning fork days when the value of comparisons between air conduction and bone conduction hearing was very well recognized. The level of interest current 50 or more years ago is well illustrated by the fact that at that time Quix was able to quote well over 200 papers concerned with bone conduction hearing, and for the most part our interest has diminished very little since the advent and development of the electrical audiometer. Some accept bone conduction audiometric test results at their face value; others emphasize their value and accuracy; and there are a few otologists and audiologists, perhaps the more discriminating, who have no confidence at all in such measurements, and who have little or nothing to do with them.

The almost universal practice of carrying out bone conduc-

*This paper has been published in full in Archives of Otolaryngology, 66:281-298, 1957.

tion tests with the receiver applied to the mastoid process of the tested ear is perhaps unfortunate, because it encourages the false notion that the opposite or untested ear remains in splendid isolation, separated from the test tone just as effectively as is recognized to be the case in air conduction tests; however, the inadequacy of bone conduction isolation has now been accepted, and the resulting and almost invariable need for masking is well recognized; but we still adhere to the mastoid process, a site with very little to recommend it.

Barany, many years ago suggested, with clearly stated reasoning, that the mastoid process was far from being the best site of application for the bone conduction receiver in clinical tests. All of our present measures of bone conduction hearing are confused to some extent by reflections of middle ear function, an inevitable result of inertia bone conduction hearing and other factors; of all the possible sites available for a bone conductor receiver, the mastoid process will give us the greatest confusion, because at this site inertia bone conduction is at a maximum. Barany also pointed out that clinical bone conduction hearing will increase as the receiver is moved closer to or into contact with the cartilage of the external ear, another argument against the adequacy of the mastoid site. There are numerous other disadvantages entailed in using the mastoid process; mastoids vary widely in tissue density and "the most sensitive spot of the mastoid" varies in position from individual to individual. Otologists who have carried out bone conduction tests will agree that it is often impossible, using a double headset with an air conduction and a bone conduction receiver, to persuade the bone conduction vibrator to do more than perch precariously behind the listener's ear; and the choice of application site disappears when the receiver can be made to stay on the head in only one position. Another difficulty encountered is reproduction of original test conditions, in terms of application site and pressure, as tests are repeated from time to time. A final objection to the mastoid type of bone conduction test is that the tested ear must remain exposed to any air-borne ambient noise during the test, because our orthodox air conduction receivers are too large to permit the simultaneous

use of both air conduction and bone conduction receivers on the same ear.

Other observations by Barany led to the conclusion that we may do better in clinical bone conduction tests if we place the bone conduction receiver on the frontal bone; inertia and cartilage conduction would thus be reduced or abolished; there would probably be less inter-subject variation in bony contour, and it is far simpler to apply a bone conduction receiver to the frontal bone than to the mastoid, and far easier to be sure of reproducing the test conditions as tests are repeated. One distinct advantage of the frontal bone site is the ever-present reminder that the ears are being stimulated equally, and that masking is almost always required; moreover, while using a frontal bone conduction receiver the two ears may be covered by air conduction receivers, and masking tones may very conveniently be fed to either ear at will.

One minor and readily overcome disadvantage of the frontal site is that at most test tone frequencies more power is required to reach the threshold of hearing than is required at the mastoid bone.

The importance of the size of the frontal sinuses in bone conduction measurements of this type remains an uncertain quantity but it is one that must be examined further.

If we are to consider seriously the possibility of making bone conduction measurements at the frontal bone while our listeners wear a pair of air conduction receivers, we must learn a good deal more than is generally known at present concerning the occlusion effect in pathological ears. We must in particular know whether cases with pathological hearing will show occlusion effects resembling those shown by the normal ear. Such evidence as there is, suggests that the normal occlusion effect will be reduced in the case with a pathological middle ear by an amount equal to the magnitude of that ear's conduction deafness.

In an attempt to gain further information on this and other related problems, about 500 ears were tested, audiometrically; some had visible middle ear changes of various types, some had otosclerosis, and some had neither visible changes nor

otosclerosis. A frontally applied bone conduction receiver was used throughout, and all the cases were tested in each of two ways: first, bone conduction with unoccluded or open ears; and second, bone conduction while the ears were occluded with a pair of standard air conduction receivers. Masking was used in adequate amount to separate the function of the two ears. The differences between the unoccluded and the occluded bone conduction thresholds were accepted as occlusion effects, and it became clear that most of the cases with normal middle ears, whether deaf or otherwise, showed the expected occlusion effect of about 15 db. at 250 and 500 c/s, a lesser occlusion effect at 1000 c/s, and no occlusion effect at 2000 and 4000 c/s.

It also became clear that the majority of the cases with middle ear changes showed no occlusion effects at *any* test tone frequency, irrespective of the size of the air conduction loss present. One further observation was that at some test tone frequencies the cases with middle ear lesions and moderate air conduction hearing losses showed bone conduction thresholds better, on the average, than the unoccluded normal ears' thresholds. In other words these observations demonstrated the presence, in cases of mild middle ear deafness, of what has been referred to for very many years as "prolonged bone conduction hearing." It should be added that the phenomenon has often been referred to as "the *illusion* of prolonged bone conduction."

It has been pointed out above that the cases with moderate middle ear deafness had better than normal bone conduction hearing, and that these same cases will show no further improvement in bone conduction hearing when their ears are occluded; but the normal ear's bone conduction hearing will improve when the ear is occluded and comes up to the level of the pathological cases with "prolonged bone conduction." There is, therefore, some justification for stating that the pathological middle ears are behaving as though they were in a permanently occluded state, a state the author has tentatively labelled "built-in-occlusion." This may be a misleading term, and comments must be limited to the observations that the effects are very similar in terms of frequency distribution and of magnitude.

The points mentioned lead to the conclusion that clinical bone conduction audiometry, carried out with occluded ears and with a frontal bone conduction receiver, is well worth serious consideration. Our orthodox techniques resemble efforts to compare the weights of a number of men, any of whom may be concealing 15 db. of lead about their persons. Occluded frontal bone tests will ensure that, whether "built-in occlusion effect" is present or absent, we derive a far less inaccurate measure of cochlear reserve.

The observations described were made in the course of a series of small scale probes into the question of clinical bone conduction hearing. The results suggest that well planned attacks on the problem are likely to be rewarding.

PROPOSED AUDIOLOGY CENTRE OF THE NATIONAL CENTRE FOR THE DEAF IN INDIA.

(Abstract).

C. A. AMESUR,
Bombay, India,
and
C. SATYANARAYANA,*
Madras, India.

For the past few years it has been the earnest endeavor of the Government of India of finding ways and means to quicken the pace of rehabilitation of the handicapped persons. With this object in view, the Committee on Deafness was appointed in 1952, and the Deaf Seminar held in September, 1955, which was ably presided by Shri U. A. Basrurkar.

Dr. C. A. Amesur, who has published a paper on "Lip Reading" in I. J. O., October, 1950, and "Rehabilitation of the acoustically handicapped" in the I. J. O., December, 1952, was requested by the Ministry of Education to give a talk on "Recent Progress in the Treatment and Prevention of Ear

*Stanley Medical College, Madras, India.

Diseases" at the Deaf Seminar, published in I. J. O., December, 1955. The Seminar accepted his following resolutions:

1. That a model Audiology Centre be set up by the Government of India;

2. That smaller audiological units should be attached to all teaching hospitals;

3. That rehabilitation of laryngectomized patients be undertaken.

On October 5, 1955, Government of India established the National Advisory Council for the Education of the Handicapped (Resolution No. F., 1-34-55, D-2, dated January 9, 1955), and nominated the author on that body.

The Council, at its first meeting, held in October, 1955, in the Council Hall of the Lok Sabha, elected the author on its Standing Committee.

The Co-author worked out the cost of such a centre, which the author placed before the Council, which accepted the same, on the following day. The cost of construction and an appropriate annual budget will be discussed.

The authors acknowledge their debt to many colleagues in Europe, the United Kingdom and the United States.

DISCUSSION.

H. W. EWERTSEN, Copenhagen, Denmark: In Denmark we now have six years' experience with the State Hearing Rehabilitation Centres, of which there are three, in cooperation with a special children's clinic. Every Danish inhabitant, with hearing troubles of chronic character, is allowed free admission to the rehabilitation program, including examination by otologist, acoustic evaluation by pure tone and speech audiometry, fitting of hearing aids, vocational guidance, teaching and follow-up.

The experience of the Copenhagen Hearing Center shows beyond doubt the necessity of a current control of all hearing aids supplied to patients. Official authorization based on a few selected specimens proved quite inadequate, providing a

false security. In Denmark the purchase of hearing aids is based on tenders in order to stimulate competition. The individual hearing aids are controlled by an "artificial ear" at the Hearing Center; the plotted frequency curves are filed, and the individual hearing aids registered on index cards so as to have detailed account of the commonest defects. Based on this file of defects, standards are prepared for improvements in new types of hearing aids. The fitting of the hearing aid is based on a close cooperation between engineers and physicians.

The capricious problem in the matter of fitting hearing aids is training in the use of the apparatus. It is of the greatest importance that the patients persevere for the first month until they have adapted themselves to the aid and its secondary noises. All the patients are referred to courses of up to four hours' instruction, and more than 50 per cent of the patients are trained in lipreading, hearing training, and speech correction. Through reports of the 63 teachers of patients with defective hearing, operating in the area of the Hearing Centers, we are able to check the correctness of the rehabilitation process. Particularly difficult cases are referred, and admitted to the college for hard-of-hearing adults at Fredericia, a rather unique institution, unknown in most other countries. The training of children is supervised by traveling teachers who visit the individual homes of the children about eight times a year. When necessary, vocational guidance is instituted.

A re-examination of the first 8,000 patients of the Hearing Center in Copenhagen showed that only 4 per cent of the patients failed to use their hearing aids. The re-examination was based on questionnaires, inquiries of personal friends, and reports from the teachers. The small figure should hence be considered reliable, and be taken as an indication of the method applied by the Hearing Center, and classification of the patients. Seventeen per cent of the applicants are not provided with hearing aids, either because their deficiency is not sufficiently pronounced or because they suffer from types of defective hearing which are not remedied by hearing

aids. Persons, who on account of mental insufficiency are unable to manage the hearing aid, are not supplied.

As compared with all other medical care, the audiological rehabilitation of the hard-of-hearing persons must be considered an inexpensive and efficient measure, which often totally changes the entire outlook on life of the patient.

INTERNATIONAL CONFERENCE ON AUDIOLOGY.

Members of the Conference:

ARGENTINA

Luciano Rodrigues Echandia, Mendoza.
Renato Segre, Buenos Aires.
Juan Manuel Tato, Buenos Aires.

AUSTRALIA

R. N. Reilly, Adelaide.

BELGIAN CONGO

John A. Elleboudt, Elisabethville.

BELGIUM

Francois Crabbe, Brussels.
Paul Hennebert, Brussels.

BRAZIL

Geraldo De Sá, Recife.
Jorge Hirschmann, Sao Paulo.
Ermiro E. De Lima, Rio de Janeiro.
Roberto Marinho, Rio de Janeiro.
Arthur Moura, Recife.
Raphael da Nova, Sao Paulo.
Mauro Penna, Rio de Janeiro.
J. E. de Rezenda Barbosa, Sao Paulo.
Mauro Candido de Souza Dias, Sao Paulo.
Nelson Venturini, Rio de Janeiro.

CANADA

William E. Goodman, Toronto.
Hollie E. McHugh, Montreal.

CHILE

Max Fontaine, Valparaiso.
J. S. Riesco Mac-Clure, Santiago.

CUBA

Jorge de Cardenas, Havana.

CZECHOSLOVAKIA

Joseph Kiml, Prague.
A. Precechtel, Prague.

DENMARK

H. W. Ewertsen, Copenhagen.
K. Faaborg-Andersen, Copenhagen.
Niels Riskaer, Gentofte.

FINLAND

Otto H. Meurman, Helsinki.
Tauno Palva, Turku.

FRANCE

Georges F. Greiner, Strasbourg.
Andre Lucas, Paris.
Guy Perdoncini, Nice.

GERMANY

Georg Heise, Berlin.
Otto Steurer, Hamburg.
H. Wullstein, Wurzburg.

GREAT BRITAIN

Sidney Allinson, Boston.
Terence Cawthorne, London.
Kenneth Harrison, Manchester.
Gavin Livingstone, Oxford.
A. Edwin Stevens, London.
Ivan Alexis Tumarkin, Liverpool.

INDIA

C. Satyanarayana, Madras.

IRAQ

M. Shawaf, Baghdad.

IRELAND

Brian O'Brien, Dublin.

ISRAEL

Rudolph Werth, Tel Aviv.

ITALY

Michele Arslan, Padua.
Ettore Bocca, Milan.
A. C. Holland, Milan.
E. Pirodda, Cagliari.

JAPAN

Shigemi Fujisaki, Osaka.
Minoru Muta, Osaka.
Fumio Nakamura, Kyoto.

MALTA

W. A. Sultana, Sliema.

MEXICO

Pedro Berruecos, Mexico City.

NETHERLANDS

J. J. Groen, Utrecht.
H. A. E. van Dishoeck, Leyden.
Henk C. Huizing, Groningen.

PERU

Ernesto Raffo, Lima.

POLAND

Jon Miodonski, Krakow.
Jerzy Szpunar, Krakow.
Josef Taniewski, Stettin.

PUERTO RICO

M. Alonso, Santurce.
Juan H. Font, San Juan.

SWEDEN

Lennart Gisselsson, Lund.
Y. Jungert, Stockholm.

SWITZERLAND

Franz Escher, Berne.

VENEZUELA

Saul Moreno, Caracas.

UNITED STATES

Bernard M. Anderman, Washington, D. C.
J. L. Bangs, Houston, Texas.
Mrs. Tina Bangs, Houston, Texas.
Fred E. Barron, Pittsburgh, Pa.
Mrs. Margaret R. Becker, St. Louis, Mo.
Willem A. van Bergeijk, Murray Hill, N. J.

UNITED STATES

Moe Bergman, New York, N. Y.
Robert C. Bilger, St. Louis, Mo.
Lester A. Brown, Atlanta, Ga.
W. T. K. Bryan, St. Louis, Mo.
Clarice Bryant, Chicago, Ill.
Joseph A. Butler, Chicago, Ill.
Peter Campanelli, Chicago, Ill.
Norton Canfield, New Haven, Conn.
Raymond Carhart, Evanston, Ill.
Claire Cooper, Nashville, Tenn.
Mary R. Costello, Detroit, Mich.
James B. Costen, St. Louis, Mo.
Walter P. Covell, St. Louis, Mo.
J. R. Cox, Jr., St. Louis, Mo.
Norman W. Crane, New York, N. Y.
Catherine Lee Cubert, Chicago, Ill.
Harold Cutler, St. Louis, Mo.
Arne Darbo, Minneapolis, Minn.
Morris Davidson, St. Louis, Mo.
Hallowell Davis, St. Louis, Mo.
K. M. Day, Pittsburgh, Pa.
Bruce Deatherage, St. Louis, Mo.
A. J. Derbyshire, Detroit, Mich.
Donald G. Doehring, Pensacola, Fla.
Leo G. Doerfler, Pittsburgh, Pa.
Mrs. Marion P. Downs, Denver, Colo.
John J. Dreher, Columbus, O.
Richard H. Ehmer, New London, Conn.
Donald H. Eldredge, St. Louis, Mo.
Aubrey Epstein, Pittsburgh, Pa.
C. F. Esselbrugge, St. Louis, Mo.
Jane Farley, Chicago, Ill.
Harvey Fletcher, Provo, Utah.
Robert D. Frisina, Washington, D. C.
Frank Frueh, Indianapolis, Ind.
John H. Gaeth, Detroit, Mich.
Robert Galambos, Washington, D. C.
Warren H. Gardner, Cleveland, O.
Victor P. Garwood, Los Angeles, Calif.
Arnold A. Gerall, Lawrence, Kans.
Henning E. von Gierke, Dayton, O.
Harry N. Glick, St. Louis, Mo.
Aram Glorig, Los Angeles, Calif.
Cornelius P. Goetzinger, Kansas City, Kans.
Robert Goldstein, St. Louis, Mo.
Victor Goodhill, Los Angeles, Calif.
Allan Goodman, St. Louis, Mo.
A. Bruce Graham, Detroit, Mich.
Rufus L. Grason, West Concord, Mass.

UNITED STATES

W. Lloyd Graunke, Knoxville, Tenn.
Glenn J. Greenwood, Los Angeles, Calif.
Hildred A. Gross, Detroit, Mich.
John Guerriero, New York, N. Y.
Frederick R. Guilford, Houston, Texas.
Ursula Hahn, Chicago, Ill.
George Harbold, Pensacola, Fla.
William G. Hardy, Baltimore, Md.
J. Donald Harris, New London, Conn.
Harriet L. Haskins, Baltimore, Md.
C. Olaf Haug, Houston, Texas.
Claude S. Hayes, Atlanta, Ga.
LeRoy D. Hedgecock, Rochester, Minn.
George Herman, Ann Arbor, Mich.
A. C. Hilding, Duluth, Minn.
Ira J. Hirsh, St. Louis, Mo.
Arthur L. Holcomb, Los Angeles, Calif.
W. H. Hopmeier, St. Louis, Mo.
L. LeRoy Horne, Wilmington, Dela.
Clarence V. Hudgins, Northampton, Mass.
Robert J. Hunter, Philadelphia, Pa.
Lloyd A. Jeffress, Austin, Texas.
James F. Jerger, Chicago, Ill.
E. W. Johnson, Los Angeles, Calif.
Kenneth O. Johnson, San Francisco, Calif.
Maurice Joseph, New Orleans, La.
Mrs. Pauline Joseph, New Orleans, La.
Jacqueline Keaster, Los Angeles, Calif.
John W. Keys, Norman, Okla.
Elizabeth C. Kimble, Atlanta, Ga.
Rhoda H. Kimmel, Minneapolis, Minn.
Robert B. King, St. Louis, Mo.
Darwin M. Knapp, Wilmington, Dela.
Ralph W. Knewitz, East St. Louis, Ill.
Heinrich G. Kobrak, Detroit, Mich.
Harriet Green Kopp, Detroit, Mich.
Lennart L. Kopra, Austin, Texas.
George Kurtzrock, Gainesville, Fla.
Paul J. LaBenz, Nashville, Tenn.
Helen S. Lane, St. Louis, Mo.
W. B. Larkin, Marshfield, Wis.
Frank M. Lassman, Minneapolis, Minn.
Joyce F. Lassman, Evanston, Ill.
Merle Lawrence, Ann Arbor, Mich.

UNITED STATES

William H. Lichte, Columbia, Mo.
J. C. R. Licklider, Cambridge, Mass.
Charles Lightfoot, Chicago, Ill.
John R. Lindsay, Chicago, Ill.
Edgar L. Lowell, Los Angeles, Calif.
Karl Lowy, Rochester, N. Y.
S. F. Lybarger, Pittsburgh, Pa.
Richard E. Marcus, Winnetka, Ill.
Freeman McConnell, Nashville, Tenn.
Malcolm McCoy, Winston-Salem, N. C.
M. A. McGinnis, St. Louis, Mo.
Mary McDermott, Detroit, Mich.
Ludwig Michael, Dallas, Texas.
James D. Miller, Bloomington, Ind.
James E. Miller, St. Louis, Mo.
Maurice H. Miller, New York, N. Y.
William E. Miller, Wichita, Kans.
Lucy M. Moore, East Lansing, Mich.
Clayton Mullin, St. Paul, Minn.
C. S. Mumma, Los Angeles, Calif.
Helmer R. Myklebust, Evanston, Ill.
Ralph F. Naunton, Chicago, Ill.
William D. Neff, Chicago, Ill.
Hayes A. Newby, Palo Alto, Calif.
Walker Nichols, Chicago, Ill.
Max H. O'Connell, San Antonio, Texas.
John J. O'Neill, Columbus, O.
Herbert J. Oyer, Columbus, O.
Charles D. Parker, Iowa City, Iowa.
Antares Parvuleson, Washington, D. C.
Frances Patton, Lafayette, Ind.
Miriam D. Pauls, Baltimore, Md.
Earl Peterson, Minneapolis, Minn.
David A. Pohlman, Pasadena, Calif.
Bruce Proctor, Detroit, Mich.
Geraldine M. Purcell, Detroit, Mich.
Grant Rasmussen, Bethesda, Md.
Mrs. Lorraine A. Roblee, Washington, D. C.
Jack Rosen, Cleveland, O.
Samuel Rosen, New York, N. Y.
Philip E. Rosenberg, Philadelphia, Pa.
H. Rosenberger, Cleveland, O.
Walter A. Rosenblith, Cambridge, Mass.
Alan S. Rubenstein, Springfield, Ill.
R. L. Ruggles, Cleveland, O.
Howard A. Runion, Stockton, Calif.
Georgia Rushford, Los Angeles, Calif.
Wayne Rudmose, Dallas, Texas.
Earl Schubert, Cleveland, O.

UNITED STATES

Harold F. Schuknecht, Detroit, Mich.
Martin C. Schultz, Philadelphia, Pa.
Lewis J. Schwartzkopf, Columbus, O.
Barbara J. Seelye, St. Louis, Mo.
Ben H. Senturia, St. Louis, Mo.
James L. Shapley, Iowa City, Iowa.
Andrew J. Shiner, Falls Church, Va.
Irvin Shore, St. Louis, Mo.
Bruce M. Siegenthaler, University Park, Pa.
S. Richard Silverman, St. Louis, Mo.
A. A. Simmons, St. Louis, Mo.
Sister Anna Bernadine, St. Louis, Mo.
Sister Anna Rose, St. Louis, Mo.
Sister Rose Antonia, St. Louis, Mo.
Catherine A. Smith, St. Louis, Mo.
Francis L. Sonday, Indianapolis, Ind.
M. Spencer, St. Louis, Mo.
Isabelle Starkey, Lansing, Mich.
Clayton Stevens, LaPuente, Calif.
S. S. Stevens, Cambridge, Mass.
Agnes A. Stokoe, Detroit, Mich.

UNITED STATES

Alice Streng, Milwaukee, Wis.
Ichiji Tasaki, Bethesda, Md.
Glenn Taylor, Normal, Ill.
Don C. Teas, Des Moines, Iowa.
Alfred R. Thea, Kalamazoo, Mich.
Nicholas Torok, Chicago, Ill.
Archie R. Tunturi, Portland, Ore.
J. Richardson Usher, St. Louis, Mo.
John A. Virant, St. Louis, Mo.
Theo E. Walsh, St. Louis, Mo.
W. Dixon Ward, Pensacola, Fla.
L. A. Watson, Minneapolis, Minn.
Harold Weber, Denver, Colo.
John C. Webster, San Diego, Calif.
Charlotte G. Wells, Columbia, Mo.
Ruedi Werth, New York, N. Y.
Edgar D. Wipperman, Covington, Ky.
Frank B. Withrow, St. Louis, Mo.
Mrs. Margaret Withrow, St. Louis, Mo.
Doris Surles Woolsey, St. Louis, Mo.
Donald M. Wright, Quincy, Ill.
Herbert N. Wright, Chicago, Ill.
Norman E. Wynn, Chicago, Ill.
Phillip A. Yantis, Ann Arbor, Mich.
Josef Zwislocki, Cambridge, Mass.

PROGRAM OF THE AMERICAN OTOLOGICAL SOCIETY, INC.

Fairmont Hotel, San Francisco, Calif., May 17-18, 1958.

Anatomical Structure of the Stapes and the Relation of the Stapedial Footplate to Vital Parts of the Otic Labyrinth.
Barry J. Anson, M.D., and Theodore H. Bast, M.D.

Repair and Consequences of Trauma to the Stapes and Oval Window in Experimental Animals.
Richard J. Bellucci, M.D., and Dorothy Wolff, M.D.

The Action of the Sense Organ of the Cochlea, Particularly from the Point of View of Neurophysiology and the Excitation of Nerve Impulses.....Hallowell Davis, M.D.

Observations on Temporary Threshold Shift Resulting from Noise Exposure.....Aram Glorig, M.D.

Congenital Stapes Fixation.....Howard House, M.D.

Physiological Mechanisms in Stimulation Deafness.
Merle Lawrence, M.D.

No Subject Assigned.....John R. Lindsay, M.D.

Portmortem Findings in Temporal Bones of Animals and Humans.....H. F. Schuknecht, M.D.

Studies Concerned with Tubo-tympanitis.
Ben Senturia, M.D.

Neurological Survey of the Children at Central Institute for the Deaf and Analyzed Results in Relation to Their Audiograms, Their Educational Advancement, Their Social Adjustment, etc.
R. S. Silverman, Ph.D., and Hallowell Davis, M.D.

A Study of Destructive Labyrinthotomy.
K. M. Simonton, M.D.

Further Studies on the Function of the Utricular Macula.
J. A. Sullivan, M.D., Walter Johnson, M.D., and Brydon Smith, M.D.

The Origin of Distortion in Cochlear Fluid Motion.
J. Tonndorf, M.D.

Pressures of the Labyrinthine Fluids Measured with an Electromanometer and Potential Value of These Pressures.
Francis L. Weille, M.D., and John Irwin, M.D.

The Differential Diagnosis of Meniere's Disease.
Henry Williams, M.D.

PROGRAM OF THE AMERICAN LARYNGOLOGICAL ASSOCIATION.

Fairmont Hotel, San Francisco, Calif., May 19-20, 1958.

Ground Substance.....Eugene S. Hopp, M.D. (By invitation)

Vocal Rest in Laryngeal Disease.....Mervin C. Myerson, M.D.

Management of Chronic Laryngeal Stenosis.
Paul H. Holinger, M.D.

Evidence of Laryngeal Participation in Emotional Expression: Its Relation to Hysterical Aphonia.
A. C. Furstenberg, M.D.

EXECUTIVE SESSION.

Panel Discussion.

AFTERNOON SESSION.

REHABILITATION OF THE POST-LARYNGECTOMIZED PATIENT.
John J. Conley, M.D., Moderator.

Types of Clinical Cases and Their Resultant Laryngeal, Esophageal, Pharyngeal and Neck Deformities.
Charles M. Norris, M.D.

The Vocal Therapist: Place and Contribution to the Rehabilitation Program.....Paul J. Moses, M.D. (By invitation)

Specific Discussion of Failures: Advanced and Difficult Technical Problems.....F. Johnson Putney, M.D.

Psychological Factors Determining the Success or Failure of the Rehabilitation Program.
Hayes A. Newby, Ph.D. (By invitation).

Film: Recording of Esophageal Speech by Means of the Image Intensifier.
Medical Division, North American Phillips Co., Inc.

Technical and Electronic Field.
Speaker from the Bell Telephone Laboratories.

New Operation for Short Circuiting the Tracheal Air into the Pharynx.....John J. Conley, M.D.

Tuesday, May 20, 1958

Changing Trends in the Treatment of Sinusitis.
Frederick T. Hill, M.D.

Nasal Obstruction Produced by Deformities about the Lobule.....Henry L. Williams, M.D.

Carcinoma-in-situ of the Larynx: a Ten-Year Study of its Histopathological Classification, Prognosis and Treatment.....Alden H. Miller, M.D.
and Russel Fisher, M.D. (By Invitation)

Bronchoscopic Findings in Tubercular Children.
D. E. S. Wishart, M.D.,
and J. B. Whaley, M.D. (By invitation)

PRESENTATION OF INSTRUMENTS.

Unusual Sinuses.....Fred W. Dixon, M.D.

Orbital Apex Syndrome.....Austin T. Smith, M.D.

Physiology of the Larynx Under Daily Stress (Film).
Paul G. Moore, M.D. (By invitation),
and Hans von Leden, M.D. (By invitation)

The Experimental Use of Homografts for Repair of the Cervical Trachea.....Joel J. Pressman, M.D.

PROGRAM OF THE AMERICAN LARYNGOLOGICAL, RHINOLOGICAL AND OTOLOGICAL SOCIETY, INC.

Wednesday, May 21, 1958

The Relationship of Thyroid Carcinoma in Children and Irradiation of the Thymus in Infancy.
Shirley H. Baron, M.D., San Francisco, Calif.

Discussion.....Robert R. Newell, M.D., San Francisco, Calif. (By invitation)

Middle Ear Effusion—Systemic Factors.
Francis W. Davison, M.D., Danville, Pa.

Open Discussion.

SYMPOSIUM—*Reconstructive Nasal Surgery.*

Moderator.....Ronald W. Taylor, M.D., Vancouver, B. C.

Panel.....Russell I. Williams, M.D., Cheyenne, Wyo.
Joseph H. Ogura, M.D., St. Louis, Mo.
Harold Owens, M.D., Los Angeles, Calif.

The Transseptal Intra-Nasal Dacryocystorhinostomy — A Neglected Operation (A Motion Picture).
E. R. V. Anderson, M.D., Los Angeles, Calif.

Malformations and Anatomical Variations Seen in the Middle Ear During the Operation for Mobilization of the Stapes (Candidate's Thesis).
Jack V. Hough, M.D., Oklahoma City, Okla.

Thursday, May 22, 1958

Soft Tissue Roentgenography of the Nasopharynx for Adenoids.....Joseph L. Goodman, M.D., New York, N. Y.

Open Discussion.

An Operative Technique to Prevent the Need of Postoperative Care in Radical Mastoid Surgery.
J. H. Thomas Rambo, M.D., New York, N. Y.

Open Discussion.

SYMPOSIUM—*Modern Criteria for Diagnosis and Therapy of Malignant Disease in the Upper Respiratory Tract.*

Moderator.....Jerome A. Hilger, M.D., St. Paul, Minn.

Panel.....Harold G. Tabb, M.D., New Orleans, La.
Walter P. Work, M.D., San Francisco, Calif.
John J. Conley, M.D., New York, N. Y.

Combined Laryngectomy and Neck Dissection (A Motion Picture).....Eugene L. Grandon, M.D., Iowa City, Ia. (By invitation)
Edgar S. Brintwall, M.D., Iowa City, Ia. (By invitation)

An Objective Approach to the Complaint: "A Lump in the Throat".....G. Arnold Henry, M.D., Toronto, Canada

Friday, May 23, 1958

A Clinical Look at Progress in the Evaluation of Hearing Function.....Frederick R. Guilford, M.D., Houston, Texas

An Unusual Paratoid Swelling—Its Diagnosis and Treatment.
Albert C. Furstenberg, M.D., Ann Arbor, Mich.

The Motor Activity of the Cricopharyngeus Muscle—A Laboratory Study (Candidate's Thesis).
John A. Kirchner, M.D., New Haven, Conn.

SYMPOSIUM—*Stapes Mobilization—Two Years Later.*

Moderator.....Howard P. House, M.D., Los Angeles, Calif.

Panel.....Merle Lawrence, Ph.D., Ann Arbor, Mich.
Philip E. Meltzer, M.D., Boston, Mass.
John R. Lindsay, M.D., Chicago, Ill.
Victor Goodhill, M.D., Los Angeles, Calif.
Samuel Rosen, M.D., New York, N. Y.
Clair M. Kos, M.D., Iowa City, Ia.
George E. Shambaugh, M.D., Chicago, Ill.
Edmund P. Fowler, Jr., M.D., New York, N. Y.

DIRECTORY OF OTOLARYNGOLOGIC SOCIETIES.

(Secretaries of the various societies are requested to keep this information up to date).

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. Erling W. Hansen, 90 So. Ninth St., Minneapolis, Minn.
Executive Secretary: Dr. William L. Benedict, Mayo Clinic, Rochester, Minn.
Meeting: Palmer House, Chicago, Ill.

AMERICAN BOARD OF OTOLARYNGOLOGY.

Meeting: Palmer House, Chicago, Ill.

AMERICAN BRONCHO-ESOPHAGOLOGICAL ASSOCIATION.

President: Dr. Walter Hoover, 605 Commonwealth Bldg., Boston, Mass.
Vice-President: Dr. Walter P. Work, 384 Post St., San Francisco, Calif.
Secretary: Dr. F. Johnson Putney, 1719 Rittenhouse Square, Philadelphia, Pa.
Treasurer: Dr. Verling K. Hart, 106 W. 7th St., Charlotte, N. C.
Meeting: Mark Hopkins Hotel, San Francisco, Calif., May 21-23, 1958.

AMERICAN LARYNGOLOGICAL ASSOCIATION.

President: Dr. Harry P. Schenk, 326 S. 19th St., Philadelphia 3, Pa.
Secretary: Dr. James H. Maxwell, University Hospital, Ann Arbor, Mich.
Place: Fairmont Hotel, San Francisco, Calif., May 19-20, 1958.

AMERICAN LARYNGOLOGICAL, RHINOLOGICAL AND OTOLOGICAL SOCIETY, INC.

President: Dr. Lawrence R. Boies, University Hospital, Minneapolis 14, Minn.
Secretary: Dr. C. Stewart Nash, 700 Medical Arts Bldg., Rochester 7, N. Y.
Place: Mark Hopkins Hotel, San Francisco, Calif., May 21-23, 1958.
Place: The Homstead, Hot Springs, Va., March, 1959.

AMERICAN MEDICAL ASSOCIATION, SECTION ON LARYNGOLOGY, OTOLOGY AND RHINOLOGY.

Chairman: Dr. Gordon D. Hoople, Syracuse, N. Y.
Vice-Chairman: Dr. Kenneth L. Craft, Indianapolis, Ind.
Secretary: Dr. Hugh A. Kuhn, Hammond, Ind.
Representative to Scientific Exhibit: Walter Heck, M.D., San Francisco, Calif.
Section Delegate: Gordon Harkness, M.D., Davenport, Iowa.
Alternate Delegate: Dean Lierle, M.D., Iowa City, Iowa.

AMERICAN OTOLOGICAL SOCIETY, INC.

President: Dr. Dean M. Lierle, State University of Iowa, Iowa City, Ia.
Secretary: Dr. Lawrence R. Boies, University Hospitals, Minneapolis 14, Minn.
Place: Fairmont Hotel, San Francisco, Calif., May 17-18, 1958.
Place: The Homestead, Hot Springs, Va., 1959.

AMERICAN OTORHINOLOGIC SOCIETY FOR THE ADVANCEMENT OF PLASTIC AND RECONSTRUCTIVE SURGERY.

President: Dr. Joseph Gilbert, 111 E. 61st St., New York, N. Y.
Vice-President: Dr. Kenneth Hinderer, 402 Medical Arts Bldg., Pittsburgh, Pa.
Secretary: Dr. Louis Joel Feit, 66 Park Ave., New York 16, N. Y.
Treasurer: Dr. Arnold L. Caron, 36 Pleasant St., Worchester, Mass.

AMERICAN RHINOLOGIC SOCIETY.

President: Dr. Russell I. Williams, 408 Hynds Bldg., Cheyenne, Wyo.
Secretary: Dr. Robert M. Hansen, 1735 No. Wheeler Ave., Portland, Ore.
Annual Clinical Session: Illinois Masonic Hospital, Chicago, Ill., October, 1958.
Annual Meeting: October, 1958, Chicago, Ill. (Definite time and place to be announced later).

AMERICAN SOCIETY OF FACIAL PLASTIC SURGERY.

President: Dr. Trent W. Smith, 327 East State St., Columbus 15, Ohio.
Secretary: Dr. Samuel M. Bloom, 123 East 83 St., New York 28, N. Y.
Meeting: New York, July 23, 1958; December 3, 1958, place to be announced.

AMERICAN SOCIETY OF OPHTHALMOLOGIC AND OTOLARYNGOLOGIC ALLERGY.

President: Dr. Joseph W. Hampsey, Grant Bldg., Pittsburgh 19, Pa.
Secretary-Treasurer: Dr. Daniel S. DeStio, 121 S. Highland Ave., Pittsburgh 6, Pa.
Annual Meeting: Palmer House, Chicago, Ill., October 16-17, 1958.

ASSOCIACAO MEDICA DO INSTITUTO PENIDO BURNIER—CAMPINAS.

President: Dr. Lech Junior.
First Secretary: Dr. Franco do Amaral.
Second Secretary: Dr. J. M. Queiroz Abreu.
Librarian-Treasurer: Dr. Souza Queiroz.
Editors for the Archives of the Society: Dr. Guedes de Melo Filho, Dr. Antonio de Almeida and Dr. Gabriel Pörto.
Meetings: Twice every month, first and third Thursday, 8:30 P.M.

ASOCIACION DE OTORRINOLARINGOLOGIA Y BRONCOESOFAGOLOGIA DE GUATEMALA.

Presidente: Dr. Julio Quevedo, 15 Calle Oriente No. 5.
First Vice-Presidente: Dr. Héctor Cruz, 3a Avenida Sur No. 72.
Second Vice-Presidente: Dr. José Luis Escamilla, 5a Calle Poniente No. 48.
Secretario-Tesorero: Dr. Horace Polanco, 13 Calle Poniente No. 9-D.

ASOCIACION DE OTO-RINO-LARINGOLOGIA DE BARCELONA, SPAIN.

Presidente: Dr. J. Abello.
Vice-Presidente: Dr. Luis Suñe Medan.
Secretario: Dr. Jorge Perelló, 319 Provenza, Barcelona.
Vice-Secretario: Dr. A. Pinart.
Vocal: Dr. J. M. Ferrando.

BALTIMORE NOSE AND THROAT SOCIETY.

Chairman: Dr. Walter E. Loch, 1039 No. Calvert St., Baltimore, Maryland.
Secretary-Treasurer: Dr. Theodore A. Schwartz.

BUENOS AIRES CLUB OTOLARINGOLOGICO.

Presidente: Dr. K. Segre
Vice-Presidente: Dr. A. P. Belou.
Secretario: Dr. S. A. Aranz.
Pro-Secretario: Dr. J. M. Tato.
Tesorero: Dr. F. Games.
Pro-Tesorero: Dr. J. A Bello.

CANADIAN OTOLARYNGOLOGICAL SOCIETY
SOCIETE CANADIENNE D'OTOLARYNGOLOGIE.

President: Dr. Robert T. Hayes, 42 Cobourg St., St. John, N. B.
Secretary: Dr. Donald M. McRae, 324 Spring Garden Rd., Halifax, N. S.
Meeting: Nova Scotian Hotel, Halifax, N. S., June 9-11, 1958.

CENTRAL ILLINOIS SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. G. C. Otrich, Belleville, Ill.
President-Elect: Dr. Phil R. McGrath, Peoria, Ill.
Secretary-Treasurer: Dr. Alfred G. Schultz, Jacksonville, Ill.

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY.

President: Dr. Stanton A. Friedberg, 122 So. Michigan Ave., Chicago 3, Ill.
Vice-President: Dr. Maurice Snitman, 408 So. 5th Ave., Maywood, Ill.
Secretary-Treasurer: Dr. Fletcher Austin, 700 No. Michigan Ave., Chicago 11, Ill.
Meeting: First Monday of each Month, October through May.

CHILEAN SOCIETY OF OTOLARYNGOLOGY.

President: Dr. Enrique Grünwald S.
Vice-President: Dr. Agustin Estartus.
Secretary: Dr. Marcos Chaimovich S.
Treasurer: Dr. Benjamin Kapkan K.
Director: Dr. Alberto Basterrica A.

DALLAS ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. Ludwig A. Michael, 3707 Gaston Ave., Dallas, Tex.
Vice-President: Dr. Hal W. Maxwell.
Secretary-Treasurer: Dr. Edward A. Newell, 1511 No. Beckley, Dallas 8, Tex.

FEDERACION ARGENTINA, DE SOCIEDADES DE OTORRINOLARINGOLOGIA.

Secretary of the Interior: Prof. Dr. Atilio Viale del Carril.
Secretary of Exterior: Dr. Aldo G. Remorino.
Secretary Treasury. Prof. Dr. Antonio Carrascosa.
Pro-Secretary of the Interior: Prof. Dr. Carlos P. Mercandino.
Pro-Secretary of the Exterior: Prof. Dr. Jaime A. del Sel.
Pro-Secretary of the Treasury: Dr. Jorge Zubizarreta.

FIRST CENTRAL AMERICAN CONGRESS OF OTORHINOLARYNGOLOGY.

President: Dr. Victor M. Noubleau, San Salavador.
Secretary-Treasurer: Dr. Hector R. Silva, Calle Arce No. 84, San Salvador, El Salvador, Central America.

FLORIDA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. Chas. C. Grace, 145 King St., St. Augustine, Fla.
President-Elect: Dr. Jos. W. Taylor, 706 Franklin St., Tampa, Fla.
Secretary-Treasurer: Dr. Carl S. McLemore, 1217 Kuhl Ave., Orlando, Fla.

FOURTH LATIN-AMERICAN CONGRESS OF OTORINOLARINGOLOGIA.

President: Dr. Dario.
Secretary:
Meeting:

GREATER MIAMI EYE, EAR, NOSE AND THROAT SOCIETY.

President: Dr. William B. Steinman.
President-Elect: Dr. James H. Mendel, Jr.
Secretary-Treasurer: Dr. H. Carlton Howard.
Meeting quarterly (March, May, October and December), on the second Thursday of the month, 6:30 P.M. at Urmey Hotel, Miami.

INTERNATIONAL BRONCHOESOPHAGOLOGICAL SOCIETY.

President: Dr. Jo Ono, Tokyo, Japan.
Secretary: Dr. Chevalier L. Jackson, 3401 N. Broad St., Philadephia 40, Pa., U. S. A.
Meeting: Seventh International Congress of Bronchoesophagology, Kyoto, Japan, September, 1958.

KANSAS CITY SOCIETY OF OTOLARYNGOLOGY AND OPHTHALMOLOGY.

President: Dr. Clarence H. Steele.
President-Elect: Dr. Dick H. Underwood.
Secretary: Dr. James T. Robison, 4620 J. C. Nichols Parkway, Kansas City, Mo.
Meeting: Third Thursday of November, January, February and April.

LOS ANGELES SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. Sol Rome.
Secretary-Treasurer: Dr. Max E. Pohlman.
Chairman of Ophthalmology Section: Dr. Richard Kratz.
Secretary of Ophthalmology Section: Dr. Carrall A. McCoy.
Chairman of Otolaryngology Section: Dr. Howard G. Gottschalk.
Secretary of Otolaryngology Section: Dr. Robert W. Godwin.
Place: Los Angeles County Medical Association Bldg., 1925 Wilshire Blvd., Los Angeles, Calif.
Time: 6:30 P. M. last Monday of each month from September to June, inclusive—Otolaryngology Section. 6:30, first Thursday of each month from September to June, inclusive—Ophthalmology Section.

LOUISIANA-MISSISSIPPI OPHTHALMOLOGICAL AND OTOLARYNGOLOGICAL SOCIETY.

President: Dr. H. K. Rouse, 1300 27th Ave., Gulfport, Miss.
Vice-President: Dr. A. J. McComiskey, 3420 Prytonia St., New Orleans, La.
Secretary: Dr. Edley H. Jones, 1301 Washington St., Vicksburg, Miss.
Meeting:

MEMPHIS SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

Chairman: Members serve as chairmen in alphabetical order monthly.
Secretary-Treasurer: Dr. Roland H. Myers, 1720 Exchange Bldg., Memphis, Tenn.
Assistant Secretary-Treasurer: Dr. William F. Murrah, Jr., Exchange Bldg., Memphis, Tenn.
Meeting: Second Tuesday in each month at 8:00 p.m. at Memphis Eye, Nose and Throat Hospital.

MEXICAN ASSOCIATION OF PLASTIC SURGEONS.

President: Dr. Cesar LaBoide, Mexico, D. F.
Vice-President: Dr. M. Gonzales Ulloa, Mexico, D. F.
Secretary: Dr. Juan De Dios Peza, Mexico, D. F.

MISSISSIPPI VALLEY MEDICAL SOCIETY.

President: Dr. Arthur S. Bristow, Princeton, Mo.
Secretary-Treasurer: Dr. Harold Swanberg, Quincy, Ill.
Assistant Secretary-Treasurer: Dr. Jacob E. Reisch, Springfield, Ill.

NETHERLANDS SOCIETY OF OTO-RHINO-LARYNGOLOGY.

(Nederlandsche Keel-Neus-Oorheelkundige Vereeniging.)

President: Dr. H. Navis, Sonsbeekweg 6, Arnhem.
Secretary: Dr. W. H. Struben, J. J. Viottastraat 1, Amsterdam.
Treasurer: Mrs. F. Velleman-Pinto, Jac. Obrechtstr. 66, Amsterdam.

NORTH CAROLINA EYE, EAR, NOSE AND THROAT SOCIETY.

President: Dr. J. C. Peele, Kinston Clinic, Kinston, N. C.
Vice-President: Dr. George E. Bradord, Winston-Salem, N. C.
Secretary-Treasurer: Dr. J. D. Stratton, 1012 Kings Drive, Charlotte 7, N. C.
Meeting:

NORTH OF ENGLAND OTOLARYNGOLOGICAL SOCIETY.

President: Mr. G. L. Thompson, 16 Ramshill Road, Scarborough, Yorkshire.
Vice-President: Mr. J. H. Otty, Frizley Old Hall, Frizinghall Road, Bradford, Yorkshire.
Secretary and Treasurer: Mr. R. Thomas, 27 High Petergate, York, Yorkshire.

OTOSCLEROSIS STUDY GROUP.

President: Dr. Joseph A. Sullivan, 174 St. George St., Toronto 5, Canada.
Secretary-Treasurer: Dr. Arthur L. Juers, 611 Brown Bldg., Louisville, Ky.
Meeting: Palmer House, Chicago, Ill.

PACIFIC COAST OTO-OPHTHALMOLOGICAL SOCIETY.

President: H. Leroy Goss, M.D., 620 Cobb Bldg., Seattle 1, Washington.
Secretary-Treasurer: Homer E. Smith, M.D., 508 East South Temple, Salt Lake City, Utah.
Meeting:

PAN AMERICAN ASSOCIATION OF OTO-RHINO-LARYNGOLOGY AND BRONCHO-ESOPHAGOLOGY.

President: Dr. Jose Gros, Havana, Cuba.
Executive Secretary: Dr. Chevalier L. Jackson, 3401 N. Broad St., Philadelphia 40, Pa., U. S. A.
Meeting: Sixth Pan American Congress of Oto-Rhino-Laryngology and Broncho-Esophagology.
Time and Place: Brazil, 1958.

PHILADELPHIA LARYNGOLOGICAL SOCIETY.

President: Dr. Chevalier L. Jackson.
Vice-President: Dr. John J. O'Keefe.
Treasurer: Dr. Joseph P. Atkins.
Secretary: Dr. Louis E. Silcox.
Historian: Dr. Herman B. Cohen.
Executive Committee: Dr. Harry P. Schenck, Dr.; Benjamin H. Shuster, Dr. William A. Lell, Dr.; William J. Hitschler.

PORTUGUESE OTORHINOLARYNGOLICAL SOCIETY.

President: Dr. Albert Luis de Mendonca.
Secretary: Dr. Antonio da Costa Quinta, Avenida, de Liberdale 65, 1° Lisbon.

PUGET SOUND ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. Clifton E. Benson, Bremerton, Wash.
President-Elect: Dr. Carl D. F. Jensen, Seattle, Wash.
Secretary: Dr. Willard F. Goff, 1215 Fourth Ave., Seattle, Wash.

RESEARCH STUDY CLUB OF LOS ANGELES, INC.

Chairman: Dr. Orrie E. Ghrist, 210 N. Central Ave., Glendale, Calif.
Treasurer: Dr. Norman Jesberg, 500 So. Lucas Ave., Los Angeles 17, Calif.
Otolaryngology: Dr. Russell M. Decker, 65 N. Madison Ave., Pasadena 1, Calif.
Ophthalmology: Dr. Warren A. Wilson, 1930 Wilshire Blvd., Los Angeles 57, Calif.
Mid-Winter Clinical Convention annually, the last two weeks in January at Los Angeles, Calif.

SECTION OF OTOLARYNGOLOGY OF THE MEDICAL SOCIETY OF THE DISTRICT OF COLUMBIA.

Chairman: Dr. J. L. Levine.
Vice-Chairman: Dr. Russell Page.
Secretary: Dr. James J. McFarland.
Treasurer: Dr. Edward M. O'Brien.
Meetings are held the second Tuesday of September, November, January, March and May, at 6:30 P.M.
Place: Army and Navy Club, Washington, D. C.

SCOTTISH OTOLARYNGOLOGICAL SOCIETY.

President: E. A. M. Connal, 1 Royal Crescent, Glasgow C. 3, Scotland.
Secretary-Treasurer: Dr. J. F. Birrell, 14 Moray Place, Edinburgh.
Assistant Secretary: Dr. H. D. Brown Kelly, 11 Sandyford Place, Glasgow.

SOCIEDAD COLUMBIANA DE OFTALMOLOGIA Y OTORRINOLARINGOLOGIA (BOGOTA, COLUMBIA).

Presidente: Dr. Alfonso Tribin P.
Secretario: Dr. Felix E. Lozano.
Tesorero: Dr. Mario Arenas A.

SOCIEDAD CUBANA DE OTO-LARINGOLOGIA.

President: Dr. Reinaldo de Villiers.
Vice-President: Dr. Jorge de Cárdenas.
Secretary: Dr. Pablo Hernandez.

SOCIEDAD DE ESTUDIOS CLINICOS DE LA HABANA.

Presidente: Dr. Frank Canosa Lorenzo.
Vice-Presidente: Dr. Julio Sanguily.
Secretario: Dr. Juan Portuondo de Castro.
Tesorero: Dr. Luis Ortega Verdes.

SOCIEDAD DE OTORRINOLARINGOLOGIA Y BRONCOESOFAGOSCOPIA DE CORDOBA.

Presidente: Dr. Aldo Remorino.
Vice-Presidente: Dr. Luis E. Olsen.
Secretario: Dr. Eugenio Romero Diaz.
Tesorero: Dr. Juan Manuel Pradales.
Vocales: Dr. Osvaldo Suárez, Dr. Nondier Asis R., Dr. Jorge Bergallo Yofre.

SOCIEDAD DE OTO-RINO-LARINGOLOGIA, COLEGIO MEDIO DE EL SALVADOR, SAN SALVADOR, C. A.

President: Dr. Salvador Mixco Pinto.
Secretary: Dr. Daniel Alfredo Alfaro.
Treasurer: Dr. Antonio Pineda M.

SOCIEDAD ESPANOLA DE OTORRINOLARINGOLOGIA.

Presidente: Dr. D. Adolfo Hinojar Pons.
Vice-Presidente: Dr. D. Jose Perez Mateos.
Secretario General: Dr. D. Francisco Marañés.
Tesorero: Dr. D. Ernesto Alonso Ferrer.

SOCIEDAD MEXICANA DE OTORRINOLARINGOLOGIA

Havre 7—Desp. 62
Mexico 6, D. F.

Honorary President: Dr. Ricardo Tapia y Fernández.
President: Dr. Máximo García Castañeda.
Secretary: Dr. Eduardo de la Parra.
Treasurer: Dr. Guillermo Pérez Villasante.
Vocal: Dr. Rafael Pacchiano.

SOCIEDAD NACIONAL DE CIRUGIA OF CUBA.

Presidente: Dr. Reinaldo de Villers.
Vice-Presidente: Dr. César Cabrera Calderin.
Secretario: Dr. José Xirau.
Tesorero: Dr. Alfredo M. Petit.
Vocal: Dr. José Gross.
Vocal: Dr. Pedro Hernández Gonzalo.

SOCIEDAD OTO-RINO-LARINGOLOGIA DE LOS HOSPITALES DE MADRID.

Presidente: Dr. Don Fernando Beltrán Castillo.
Secretario General: Dr. Don Alfonso Vassallo de Mumbert.
Tesorero: Dr. Don Rafael García Tapia.

SOCIEDAD VENEZOLANA DE OTORRINOLARINGOLOGIA.

Presidente: Dr. Alfredo Celis Pérez.
Vice-Presidente: Dr. Bustamante Miranda.
Secretario General: Dr. Jesús Miralles.
Tesorero: Dr. M. Matheus.
Vocales: Dr. Perez Velasquez and Dr. Wilmer Palacios.

SOCIEDADE DE OFTALMOLOGIA E OTORRINOLARINGOLOGIA DO RIO GRANDE DO SUL.

President: Dr. Paulo Fernando Esteves.
Vice-President: Dr. Jayme Schilling.
First Secretary: Dr. Carlos Buede.
Second Secretary: Dr. Moizés Sabani.
First Treasurer: Dr. Israel Scherman.
Second Treasurer: Dr. Rivadávia C. Meyer.
Librarian: Dr. Carlos M. Carrion.

SOCIEDAD PANAMENA DE OTORRINOLARINGOLOGIA

Presidente: Dr. Manuel Preciado.
First Vice-Presidente: Dr. Alonso Roy.
Second Vice-Presidente: Dr. Carlos Arango Carbone.
Secretario: Dr. María Esther Villalaz.
Tesorero: Dr. Ramòn Crespo.

SOCIEDADE PORTUGUESA DE OTORRINOLARINGOLOGIA E DE BRONCO-ESOFAGOLOGIA.

Presidente: Dr. Alberto Luis De Mendonca.
Vice-Presidente: Dr. Jaime de Magalhaes.
1.° Secretario: Dr. Antonio da Costa Quinta.
2.° Secretario: Dr. Albano Coelho.
Tesoureiro: Dr. Jose Antonio de Campos Henriques.
Vogais: Dr. Teofilo Esquivel.
Dr. Antonio Cancela de Amorim.
Sede: Avenida da Liberdade, 65, 1°, Lisboa.

SOCIETY OF MILITARY OTOLARYNGOLOGISTS.

President: Capt. William C. Livingood, U.S.N. (M.C.)
Secretary-Treasurer: Lt. Col. Sanley H. Bear, M.C., 3810th USAF Hospital, Maxwell AFB, Alabama.
Meeting: Palmer House, Chicago, Ill.

SOUTH CAROLINA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. James H. Gressette, Orangeburg, S. C.
Vice-President: Dr. Robert P. Jeanes, Easley, S. C.
Secretary-Treasurer: Dr. Roderick Macdonald, 333 East Main St., Rock Hill, S. Car.
Meeting:

SOUTHERN MEDICAL ASSOCIATION, SECTION ON OPHTHALMOLOGY AND OTOLARYNGOLOGY.

Chairman: Dr. V. Eugene Holcombe, Charleston, W. Va.
Chairman-Elect: Dr. G. Slaughter Fitz-Hugh, Charlottesville, Va.
Vice-Chairman: Dr. George M. Haik, New Orleans, La.
Secretary: Dr. Mercer G. Lynch, New Orleans, La.

VIRGINIA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. Benjamin Sheppard, 301 Medical Arts Building, Richmond, Virginia.
President-Elect: Dr. Emanuel U. Wallerstein, Professional Building, Richmond, Virginia.
Vice-President: Dr. Calvin T. Burton, Medical Arts Building, Roanoke, Virginia.
Secretary-Treasurer: Dr. Maynard P. Smith, 600 Professional Building, Richmond, Virginia.
Meeting:

WEST VIRGINIA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. James K. Stewart, Wheeling, W. Va.
Secretary-Treasurer: Dr. Frederick C. Reel, Charleston, W. Va.
Annual Meeting: Greenbrier, White Sulphur Springs, W. Va., May 31st through June 1st.